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SCIENTIFIC PAPERS

CHEMOTHERAPY OF LEUKEMIA[1]

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The chemotherapy of leukemia has made progress since the first use, in 1865, of arsenic in the treatment of two cases of chronic leukemia. Although radiation remains the most used therapy for the chronic leukemias, most recent progress has been made in chemotherapy.

Some of the real advances in this discipline actually lie in the theoretical realm, rather than in practical therapy.

Neither radiation nor presently available chemotherapy cures any type of leukemia. The clinician can, however, prolong the lives of many patients with acute leukemia and, in the chronic forms at least, the useful life and perhaps even the survival time of patients can be increased.

There are several classifications of agents which are useful. I will mention them briefly now and return to them in going through the individual types.

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Aminopterin, Methotrexate, Meticorten, Myleran, Triethylene melamine and Urethane are trade names.

The antimetabolites have been most useful in acute leukemia. Among these we number the antagonists of folic acids and purine analogues, and a newer group of agents which are probably acting as glutamine antagonists.

The adrenal steroid hormones have proven to be very useful in acute leukemia and occasionally useful in the chronic leukemias. The polyfunctional alkylating agents, that is agents such as nitrogen mustard and those which act similarly, will be mentioned as well as the miscellaneous groups which come under the heading of general cell poisons.

Acute Leukemia

In acute leukemia, what we aim for is a remission. We use both supportive and specific therapy. Supportive therapy of course includes transfusions, antibiotics when indicated, and intravenous fluids. These, in themselves, will not produce a true remission, although they may produce some subjective improvements. A true remission is one in which the marrow returns to or toward normal and, in most cases, this is accompanied by clinical improvement; improvement in peripheral blood findings; decrease in

the size of liver, spleen, nodes or other masses when present. Such a remission rarely occurs spontaneously.

The marrow aspirate of a child with acute leukemia shows a monotonous picture of undifferentiated blast cells with some mitoses. There are few or no active erythroid elements in such marrows, no megakaryocytes, and no platelets. The marrow in adults with acute leukemia is often somewhat different. The blastic picture is not as common. They more often have some differentiated cells, but functionally these cells do not act as mature granulocytes.

When a remission has been produced, megakaryocytes appear. These produce platelets. Normal erythroid elements, mature granulocytes, and other elements of the myeloid series are present. When this happens, as aforesaid, we usually have concurrent clinical and peripheral blood improvement. We aim for this with one of three groups of compounds—the folic acid antagonists, purine antagonists, or the adrenal steroids.

The folic acid antagonists were first shown to be of benefit in acute leukemia by Farber, in 1948. Aminopterin and Methotrexate (Amethopterin) are four amino substituted antagonists of folic acid and are the most clinically useful of this group of compounds. They are almost always given orally. The average dose for a five-year-old child is 0.25 to 0.5 mg. of Aminopterin, and 2.5 to 5.0 mg. of Methotrexate. The dose is not correspondingly increased in adults. The dose of Methotrexate in adults usually starts at 5 mg. a day. Since there is approximately a ten-fold difference in the dosage of these two drugs, it is very important to differentiate between the two. Caution must be used in treating patients with impaired renal function since, in this situation, considerable amounts of the antagonist will remain in the serum for prolonged periods after administration. At least the initial remission can usually be obtained in children with normal renal function without the development of toxicity.

Eventually, however, as with any of the agents we use, the leukemic cells develop resistance to these antagonists. The normal cells don't develop such resistance and at a point at which there may be some effects on the abnormal cells, toxic signs appear. If the drug is stopped at the time that anorexia and mouth ulcerations appear, the other signs of toxicity (diarrhea, intestinal ulcerations) do not occur and no danger to the patient ensues. If mouth ulcers develop, treatment is discontinued temporarily and is resumed after the signs of toxicity subside and is continued until the remission is achieved.

It frequently takes more than one course of treatment to produce a remission and eight weeks or more of treatment may be necessary to obtain a remission. The average length of such remissions in children is approximately eight months, varying in individual cases from one month to over two years. Good clinical and hematological remissions occur in 30 to 50 per cent of the children treated with folic antagonists. Adults—and by that I mean anyone over the age of 14 or 15—do not respond to the folic acid antagonists. Probably fewer than 10 per cent (or even less in some series) respond.

Methotrexate has also been found to cause improvement, after intrathecal administration, of central nervous system involvement with leukemia. This has been true even in children whose disease generally is resistant to the drug. The greater effect is presumably due to the higher concentration of Methotrexate in the spinal fluid.

6-Mercaptopurine is a completely different type of antimetabolite. It probably acts at a different metabolic site, although this, like the folic acid analogues, is presumed to act at some point in the course of nucleic acid synthesis. Of the purine analogues, 6-mercaptopurine is the most widely used, although 6-chloropurine and thioguanine are equally useful clinically. 6-Mercaptopurine is given orally to children or adults with a daily dose of 2.5 mg./kg. body weight. From three to twelve weeks may be necessary for beneficial effects to be noted. At this dose level in leukemic patients, toxic effects are rare

in children. We do see occasional nausea and anorexia in adults. The only toxicity is usually a decrease in the white count and actually some decrease is necessary before improvement occurs, although the fall in white count *per se* is not a therapeutic response.

It is important to note many of these patients when first seen are leukopenic and, of course, the majority of them are thrombocytopenic. This is not a contraindication to the use of the antimetabolites.

As remission develops (as evidenced by a decrease in the abnormal cells and an increase in the erythroid elements in the marrow), the leukocyte and platelet levels rise to normal despite continued therapy with agents that are bone marrow depressants in normal patients.

6-Mercaptopurine produces remissions in approximately one-third of the children and fewer of the adults—about one-fifth of the adults so treated. Remissions in children occur in those whose disease is resistant to Methotrexate and cortisone as well as in previously untreated cases of acute leukemia. Thus, there is no cross resistance between these two groups of agents. Since at least three weeks may elapse before beneficial effects result from the use of a folic acid antagonist or the purine analogues, the adrenal cortical steroids or ACTH, are the treatment of choice when quick effects are necessary. We feel that such rapid action is necessary when the patient develops severe bleeding, high fever, or is generally getting worse very rapidly.

The anti-leukemia effects of the different adrenal cortical steroids do not differ, but the side effects do. Meticorten is used widely now because of the lack of salt retention. It is very important to use adequate doses. What we consider adequate doses of the steroids are considered large doses in other fields. In children we use at least 100 mgs. of cortisone while adults receive 300 mgs. daily, orally. We don't usually give it intramuscularly for two reasons. Patients tend to bleed and intramuscular injections are therefore avoided. Also intramuscular cortisone does not provide the rapid effects desired. Using hydrocortisone, the dose is about two-thirds of the above orally or intravenously. The dose of Meticorten used for children is 50 mgs. a day and for adults 100 mgs. a day. If the patient is acutely ill and bleeding and cannot take oral medication, ACTH intravenously is sometimes of benefit. It is not given as a single dose but as around-the-clock medication, 25 to 50 mgs. a day throughout the twenty-four hour period. Doses of a gram of Meticorten have been used and an increased number of responses have been reported. We have not been able to repeat such results. We don't find any greater effect at a gram than at 100 mgs. The adrenal cortical steroids produce remissions in children about 50 to 70 per cent of the time. About 25 per cent of the young adults so treated have remissions. However over the age of thirty, a remission due to an adrenal steroid is very rare. All of the remissions from steroids are usually of very short duration.

Since the development of resistance influences profoundly the results obtained in the chemotherapy of leukemia, many investigators connected with the field of genetics, biochemistry and chemotherapy of infectious disease feel that combination therapy (similar to that used in the treatment of tuberculosis) might well prevent the development of this resistance and answer the therapeutic problem of leukemia. To date many combinations have been tried with no increased benefits.

Azaserine, another antimetabolite, appears to be acting as a glutamine antagonist and by virtue of this causes inhibition of nucleic acid biosynthesis. Azaserine, or the related compound diazooxonorleucine (which we call DON, and which acts similarly) when given alone are of no practical benefit in the treatment of leukemia. These compounds can cause only very brief partial remissions. However, when a group of children were treated with a combination of 6-mercaptopurine and azaserine, there seemed to be some benefit above that obtained with 6-mercaptopurine alone.

The increase was not so much in the incidence of the remissions but in the duration of the remissions, and this combination is now being studied further by cooperative groups throughout the country. There is randomized selection of cases for comparative therapy to see whether there actually is such an increase in the duration of remission. The preliminary data seem to indicate that this is so. In a small group of adults so treated the results have been no better than with 6-mercaptopurine alone.

X-ray therapy is not usually considered to be the treatment for acute leukemia, and some feel that it should be avoided at all times. There are instances when it is of use. If symptoms are produced by a tumor in a vital area such as in the central nervous system or the mediastinum resulting in symptoms due to pressure, small doses of X-ray may be of benefit. It is also useful for joint effusions and skin nodules which occasionally appear. These latter are not usually an acute problem but sometimes are painful.

In planning the treatment of patients with acute leukemia, it is necessary to take into account certain factors most of which have already been mentioned. The first thing to consider is the fact that the antimetabolites although they cause longer and more satisfactory remissions than the steroids, take from three to twelve weeks to exert their beneficial effect. The adrenal steroids and ACTH act more rapidly, but the remissions are shorter. The folic acid antagonists usually are of no benefit to adults. There is no cross resistance between the three different classes of agents. Using the above as guide posts, the sequence of therapy for an individual patient is decided. With an acutely ill patient, whether a child or an adult, an adrenal cortical steroid is used. With an adult, 6-mercaptopurine may be started as well, because there is very little hope of anything resulting from the steroid other than a temporary decrease in bleeding tendency without any change in platelets. If the condition of the child is fair to good, either Methotrexate or 6-mercaptopurine is used and the other is used subsequently when resistance to the first develops. There is no reason to choose one of these rather than the other. In adults, however, there is no point in starting with the folic acid antagonist. The possibility of obtaining a remission with 6-mercaptopurine is much greater. Occasionally adults respond to Methotrexate after resistance to 6-mercaptopurine develops. Any of these agents is continued until one is definitely sure that the patient is not responding (which may mean many weeks of treatment) or until the patient who has shown a response becomes resistant to the agent. If at any time he becomes acutely ill during the course of treatment, then a steroid is used.

If 6-mercaptopurine produces a remission, it is fairly important to continue treatment with this agent throughout the period of remission. Relapses (off therapy) are fairly rapid and the problem of the prolonged period of treatment before response again arises.

Prior to the use of any specific antileukemic therapy, only 5 per cent of the children with acute leukemia survived a year. Once the adrenal steroids and Methotrexate became available 29 per cent survived for a year, but recently with the use of Methotrexate, cortisone, 6-MP, and azaserine, (sequentially) 50 per cent have survived a year or more.

The adults have not shown any such response. When the folic acid antagonists and the adrenal steroids, but not 6-mercaptopurine, were available, 50 per cent survived six months. With the use of 6MP 50 per cent survived about six months. That isn't the whole picture however. The group that had remissions on 6-mercaptopurine or 6-chloropurine survived longer (10–12 months). Of course they are a selected group since they had lived long enough to have treatment for at least three weeks. Those who have had treatment for at least three weeks, however, and have not responded survived only six months.

Chronic Granulocytic Leukemia

When patients are seen initially with chronic granulocytic leukemia, unlike chronic lymphatic leukemia, they are frequently symptomatic. They may be anemic and usually require treat-

ment at the time when first seen. There are a number of agents available for control of this disease. The over-all course of the disease is probably not appreciably affected by therapy. The only person who has claimed prolongation of life is Osgood who uses regularly spaced titrated doses of X-ray therapy or P 32. Even without an increase in the duration of disease, proper treatment keeps patients comfortable, allowing an active working life until the last few months of life.

The treatment of choice in chronic granulocytic leukemia depends on the stage of the disease. With early disease, where the spleen is large and anemia may be mild but symptoms are few, many agents are useful. This list includes X-ray (external irradiation and P 32), polyfunctional alkylating agents (nitrogen mustard, Triethylene melamine, triethylene phosphoramide (TEPA), triethylene thiophosphoramide (Thio-TEPA), chlorambucil, Myleran, Urethane, Fowler's Solution and 6-mercaptopurine). X-ray is the most widely used form of treatment. Myleran is probably equally useful in causing clinical and hematologic improvement early in the disease. 6-MP works well at this stage but it is often reserved for later use because it may be effective at a time when the platelets are low and other agents are contra-indicated. Fowler's Solution is still a useful compound. Urethane is also effective, although the high incidence of nausea and vomiting limits its use.

As the disease progresses, larger doses of X-ray may be necessary and Myleran becomes less effective at tolerated doses. Ultimately platelets begin to fall, the patient becomes more anemic, nodes become enlarged, and fever may be a problem as the terminal blastic form of the disease develops. The previously mentioned agents are usually no longer useful and cannot be used in the presence of thrombocytopenia. Cortisone may reduce bleeding tendencies but it does not otherwise alter the disease process. 6-MP is occasionally useful in providing remissions of several months at this time.

In view of the importance of the alkylating agents in the treatment of chronic granulocytic leukemia they will be discussed in more detail. Despite the larger number of such drugs available they probably vary from one another mainly in the incidence and degree of side effects rather than in the therapeutic effects. Nitrogen mustard must be given intravenously. It usually causes nausea and vomiting which can rarely be completely controlled. However, that is no contra-indication for the use of the drug when rapid action is required. Triethylene melamine is given orally with a dose of 5 to 10 mg. weekly to a total of 20 to 40 mg. It occasionally causes nausea and anorexia and there is some variation in the sensitivity of patients so that the dose must be watched carefully. Several weeks should be allowed after each course to insure maximum hematological effect before more is given. Myleran is generally more readily tolerated without side effects and is given daily rather than intermittently. The usual daily dose is 4 to 6 mgs. It may take several weeks for the effects to occur. Once the leukocyte count reaches a normal level the drug should be discontinued since occasionally retreatment is not necessary for many months. However, most patients will have a relapse of their counts within four to five weeks. They should then receive Myleran again and, once brought under control, be maintained on a dose of one to two mgs. a day. Once the individual's pattern of response is known treatment can be regulated. Myleran can cause thrombocytopenia, especially when larger doses are used initially. The count should be checked at intervals to make sure that thrombocytopenia is not developing. If it occurs, treatment should be stopped until the platelets increase. Myleran resembles most of the agents already mentioned in this paper in being active in a dose range that is relatively close to toxicity. The doctor must be aware of the toxic side effects and watch for them, but cannot let the possibilities prevent use of the drug.

Chronic Lymphatic Leukemia

Chronic lymphatic leukemia often does not require treatment for prolonged periods. The mere presence of an elevated leukocyte level or

the presence of this and some large nodes, does not mean that the patient should be treated. He may well have a perfectly normal hemoglobin level and normal platelet count with no symptoms attached to these abnormal findings. Patients should not be treated for an elevated white count alone. However, when systemic manifestations such as weakness, weight loss, or fever become obvious; if anemia becomes a problem; or if the nodes are situated in an area that will cause trouble; then treatment is necessary. Treatment has to be started pretty cautiously with chronic lymphatic leukemia because these patients may be more sensitive than those with other diseases.

Early in the disease, if treatment is indicated small doses of X-ray are very useful. Triethylene melamine is effective but the dose is much less than that previously mentioned for chronic granulocytic leukemia. In chronic lymphatic leukemia only a single dose of 2.5 mgs. is given, since that dose alone may be enough to cause a marked and rapid fall in the white count, a fall in the platelet count and temporary marrow depletion. It is necessary to wait several weeks for the maximum effect before giving any more.

CB1348, chlorambucil is a newer alkylating agent, useful in the treatment of chronic lymphatic leukemia. It has very few side effects. The daily dose is 0.1 to 0.2 mgs./kg. body weight daily. As with other alkylating agents used in chronic lymphatic leukemia, the dose required may be low. Therefore, drug administration is started with 0.1 mg./kg. daily and the blood count is watched closely. When the count comes down to normal level or is falling rapidly, the drug is stopped.

When lymphadenopathy is present, a rapid fall in the leukocyte count and in the size of the nodes may cause hyperuricemia. This may be of such a degree that it cannot be handled by the kidneys with the consequent production of uric acid stones and possibly complete urinary obstruction. Hyperuricemia is frequently present without treatment in chronic granulocytic leukemia and in acute leukemia but not prior to treatment in chronic lymphatic leukemia. Hyperuricemia may be produced in the different types of leukemia by irradiation, polyfunctional alkylating agents, 6-mercaptopurine, and adrenal cortical steroids.

As the disease progresses, irradiation and the alkylating agents continue to be useful. Platelet depression during treatment indicates a need to discontinue therapy at least temporarily. If the platelet count is depressed before any treatment, however, and if the marrow is hypercellular, presumably the platelet count was low because of the presence of the abnormal cells. This is the time that the use of an antileukemia agent is indicated. Decreasing the abnormal cells in the marrow will allow an increase in the platelets and a decrease in the bleeding tendency.

Cortisone or the other adrenal cortical steroids are useful in one specific situation in lymphatic leukemia. Aside from the fact that they can cause a marked decrease in the leukocyte level and the size of the lymph nodes, they are specifically indicated when hemolytic anemia is present. The acquired hemolytic anemias which occasionally occur in the course of leukemia, are seen predominantly in chronic lymphatic leukemia. They are often, but not always, accompanied by a positive Coomb's test. Adrenal cortical steroids act to prevent hemolysis, allowing maintenance of hemoglobin levels. In this case the doses necessary are not those used in treating acute leukemia. Fifty mgs. of cortisone a day or less in an adult may maintain the hemoglobin level in a patient with acquired hemolytic anemia due to chronic lymphatic leukemia. If the patient is acutely ill, however, larger doses of steroid should be given initially so as to get a rapid effect and then the dose can be decreased. Patients have been maintained for several years on low doses of the adrenal steroids.

In occasional cases of chronic lymphatic leukemia administration of adrenal steroid produces an elevation of the leukocytes. This does not interfere with the beneficial effects on the hemoglobin.

As the disease progresses, bleeding manifesta-

tions become more prominent. Cortisone may stop the bleeding tendency temporarily, but it is not much benefit in the disease at the late stage.

It is necessary to stress again that the total care of the patient is particularly important in the treatment of leukemia. Specific therapy is only one aspect of the management of the patient. Massive doses of antibiotics should be employed whenever serious infections threaten the patient, but routine use of antibiotics prophylactically should be avoided, regardless of the level of the white count. The use of antibiotics promotes development of moniliasis and of infections with antibiotic resistant bacteria. The antibiotics are therefore saved for specific periods of infection. Of course leukemics are frequently febrile without explanation and one is obligated to use antibiotics, but they should not be continued for weeks at a time. Transfusions should be given as often as necessary to combat anemia. Nine or ten grams is usually an adequate level in these patients and they can receive transfusions on an outpatient basis. Thus the hospital stay is relatively short and the patient is encouraged to lead as normal a life as possible.

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DIABETES DETECTION WEEK

November 16–22, 1958

Once again the Committee on Diabetes of the Medical and Chirurgical Faculty is preparing for its yearly program, in which an endeavor is made to interest the public in visiting its family physician to be tested for Diabetes. As in the past, the Maryland Pharmaceutical Association and the Maryland Dietetic Association are cooperating in this statewide task.

In Talbot and Wicomico Counties, the Maryland Society of Hospital Volunteers is supporting programs in Easton and Salisbury in cooperation with their local Diabetes Committees.

In Salisbury, a Diabetes Detection Center, where both urine and blood will be tested, is planned for Wednesday and Thursday, November 19 and 20, and on November 21 a similar program will be carried out in Easton.

For the remainder of the state, through the Maryland Pharmaceutical Association, drug stores will give self-testing urine kits to the public, which will be returned to this Committee for analysis. All physicians are urged to give this program the same wholehearted cooperation they have in the past.

INDICATIONS FOR TRACHEOTOMY*

ADOLPH WOLFERMAN, M.D.

Between January 1, 1947 and July 1, 1950, 108 patients underwent tracheotomy at the Ochsner Foundation Hospital in New Orleans, according to a report by Echols and others. Thirty-nine of these patients each had an obstructive lesion of the upper airway. The remaining 69 patients had no laryngeal or pharyngeal pathology. They had various neurological, medical, or surgical disorders and tracheotomy was done to correct or prevent respiratory complications.

This situation is characteristic of many progressive medical centers and teaching hospitals throughout this country. It is realized more and more that tracheotomy, far from being a dangerous or heroic procedure, has saved many medical and surgical patients whose prognosis had seemed utterly hopeless. With the virtual disappearance of diphtheria and the conquest of acute laryngo-tracheo-bronchitis by antibiotics, tracheotomy for pharyngeal or laryngeal diseases has become necessary rarely. Many hospitals, however, use this procedure almost routinely in certain critically ill and comatose patients who seemingly have no sign of respiratory obstruction. Tracheotomy, in addition to relieving upper respiratory obstruction is of value in keeping the lower airways clear and in improving oxygenation.

Large amounts of fluid secreted by the pharynx, salivary glands, trachea, bronchi, and even alveoli, will accumulate in the airways of a greatly weakened or not quite conscious patient whose cough reflex is absent or reduced. Sucked into his tracheo-bronchial tree, it will finally flood his respiratory system and interfere with his air exchange to a point incompatible with life. No wonder-drug, no surgical procedure will be of any help, unless efficient respiratory exchanges are maintained. Tracheotomy gives an easy and immediate access to the lower airways. A tracheotomy can be kept patent by frequent suction and it frequently aids in the survival of a critically ill person.

This has been shown to be true in poliomyelitis. Almost all patients in respirators are tracheotomized. However, the problem of pooled secretions sucked into the lower airways and eventually leading to so-called aspiration pneumonia, exists in every patient in coma. It also exists in patients whose cough reflexes are diminished due to brain damage, cerebral edema, cerebral vascular accidents, head injuries, eclampsia gravidarum, barbiturate poisonings, neurosurgical procedures, tetanus, and others. It also exists in chest injuries and thoracic surgery, when pain makes coughing and breathing difficult. Any condition requiring large amounts of sedation and narcotics will eventually be complicated by a depressed respiratory center. In these circumstances, tracheotomy should be considered.

Obstruction of airways by pooled secretions will lead to important changes in respiratory physiology.

A decrease in air exchange causes anoxemia and later an increase of CO_2 in the blood and of lactic acid in the tissues leading to acidosis. These changes in the blood chemistry, will affect the patient's consciousness and lead to coma. Lack of consciousness will further aggravate the respiratory obstruction because of an increasing amount of pooled secretions in the upper airways and of further dulling of the cough reflex. We are dealing here with a vicious cycle. There are several other vicious cycles: Anoxemia increases capillary permeability. In the lungs, this favors the occurrence of pulmonary edema, which in turn aggravates the already existing anoxemia. There also is a direct action of oxygen lack and

* Read before the Department of Otolaryngology of the University of Maryland on March 24, 1958.

hypercapnia on the respiratory center, which after a brief period of stimulation becomes exhausted. Paralysis of the respiratory center decreases depth and rate of respiration leading to more oxygen want. Tracheotomy will instantly break these vicious cycles and relieve these grave disturbances of the normal respiratory physiology.

Tracheotomy reduces the dead space air in the upper airways from 150 cc. to 50 cc. This is an important fact in the cases of very ill patients whose vital capacity is often greatly reduced, since 100 cc. of additional fresh air is made available to the alveoli for ventilation. Breathing is made much easier since the pharyngeal pool of mucus and the pharyngeal muscles and vocal cords are by-passed. Under normal circumstances the considerable resistance of the vocal cords to air intake is not felt. However, it is well known that it is often very difficult to train a young child to return to normal breathing through the nose or mouth after it has worn a tracheotomy cannula for a long time. The child finds it much easier to breathe through the cannula than through the more resistant natural airways.

After the obstructing mucoid fluid is removed from the tracheo-bronchial tree by suction, ventilation improves, CO_2 is eliminated, and adequate oxygen is available to the tissues. The respiratory center recovers. Breathing that was noisy and labored becomes quiet and regular. Often a violent and delirious patient will become cooperative and rational.

Tracheotomy is contra-indicated when there is pulmonary edema due to cardiac decompensation. Here, obviously, respiratory obstruction is not the cause of anoxemia but its consequence. Tracheotomy and aspiration will not remove the primary pathology.

The technique of tracheotomy is adequately described in many textbooks of otolaryngology and surgery. The surgical steps are few and simple. However, a certain amount of experience is necessary in order to perform this procedure successfully.

Tracheotomy occasionally has gotten a bad reputation because of fatalities during surgery or shortly afterwards. These unfortunate accidents happen in ill-equipped emergency rooms, where untrained physicians, medical students and even nurses perform this operation in an atmosphere of utter confusion and despair—with poor lighting, inadequate anesthesia, defective instruments and often on a moribund patient. One frequently hears: "We have nothing to lose." This is a most dangerous slogan. As long as the patient is alive, we have a patient to lose. There is no place for this sort of fatalistic attitude in tracheotomy, and certainly no justification for unskilled and negligent "emergency" surgery. If we feel that the patient is lost, it is better to let him die without a tracheotomy.

The technique of tracheotomy has been revolutionized by the development of endoscopy and especially endotracheal intubation. Endotracheal intubation makes tracheotomy an orderly procedure under any circumstances. The endotracheal tube guarantees an adequate airway until the procedure is completed safely. There is no race against time. The patient can be taken to the operating room and properly prepared and oxygenated. The neck can be adequately anesthetized, sterile technique can be used, and one can wait for a competent assistant. Bleeding vessels can be leisurely ligated and if an inadvertent injury to a larger vessel occurs the accident need not cause the operator to panic. Oxygen, and even a light general anesthetic can be administered throughout the procedure. The operative field should be dry before the trachea is incised and the endotracheal tube withdrawn. Aspiration of blood into the trachea and bronchi, which is undesirable, is thus prevented. Anyone undertaking a tracheotomy should be capable of intubating, or there should be an otolaryngologist available who is skilled in endotracheal intubation. To begin a tracheotomy on a critical patient without being prepared for intubation means not giving the patient a fair chance of survival. There are, of course, cases in which intubation is impossible—such as laryngeal obstruction by tumor or foreign body—but these

cases are relatively rare. Intubation is especially desirable in young children and newborn babies where tracheotomy can be a most difficult procedure because of the small size of the trachea.

Adequate local anesthesia is an important point. Pain will make patients uncooperative and restless and will reflexly increase their anoxia through laryngospasm. The skin and deeper structures should be infiltrated over a relatively large area with an efficient local anesthetic to which a small amount of adrenaline is added. I prefer 1 per cent xylocain and use between 10 and 20 cc.

An experienced assistant, one who knows how to retract tissues and how to tie vessels, should be engaged whenever possible. Many a surgeon has lost the trachea in a confusion of soft tissues, thyroid and bleeding vessels because he could not work on the trachea and at the same time retract the tissues covering it. It is easy to understand that this difficulty occasionally proves fatal to the patient. Nothing is more annoying than to begin a tracheotomy and, after being well on the way, to discover that an important instrument is missing from the set. Adequate retractors are essential and should be included in every tracheotomy set.

Tracheotomy has become almost routine for polio patients in respirators. Any patient whose breathing mechanism is so severely affected as to require the aid of a mechanical respirator needs a tracheotomy. As previously explained, tracheotomy will eliminate the resistance of the larynx and pharynx and greatly facilitate air exchange. It also reduces the dead air space from 150 cc. to 50 cc. Occasionally, tracheotomy will improve ventilation to such an extent as to make the use of a respirator unnecessary. Progressive difficulty in swallowing, leading to accumulations of secretions in the throat—as seen in bulbar poliomyelitis, diphtheria, and many other neurological and neurosurgical conditions—makes tracheotomy imperative. The powerful iron lung may suck these secretions into the tracheobronchial tree, making tracheotomy all the more important.

The eclampsia of pregnancy is another condition in which tracheotomy has been found to be useful and, at times, life saving. Maintenance of an adequate airway is part of the basic management of eclampsia. Laryngospasm during coma and convulsions can be fatal. Hypoxia due to pooling of secretions, and laryngospasm may lead to cerebral edema and hemorrhage. Many eclamptic patients show cerebral hemorrhage at autopsy. The heavy sedation required in eclamptic patients depresses the respiratory center and abolishes cough reflex. Improved ventilation through tracheotomy will compensate to a certain extent for the depression of the respiratory center. Better oxygenation of the brain lessens the danger of convulsions and therefore less sedation is required. Collins and others report that tracheotomy has reduced the mortality rate in eclampsia from 8 per cent to 3 per cent.

Patients with barbiturate poisoning are often deeply comatose. The medullary respiratory center and the sino-aortic chemoreceptors are depressed resulting in a decrease of the depth and rate of respiration. Secondary pulmonary edema and hypostatic pneumonia are the usual complications. Death is due to respiratory failure. Large doses of barbiturates also depress the central vaso-motor center and cause peripheral vasodilatations and hypotension. Respiratory exchanges and blood pressure must be maintained. Many patients drown in their own secretions, which pool in the hypopharynx and tracheobronchial tree. In the absence of cough and gag reflexes, these patients are unable to take care of their secretions. Tracheotomy is obviously a necessity in these cases.

Much has been written on tracheotomy in head injuries. Practically every patient with a severe head injury is in danger of partial respiratory obstruction. Often breathing is so shallow that inspirations and recoil expirations only move the dead air space back and forth within the tracheo-bronchial tree. The absence of cyanosis does not necessarily indicate an adequate airway. The causes of respiratory obstruction in

head injuries are many. Attempts to swallow may lead to entry of secretions or food into the larynx, which is unable to expel the foreign matter because of absent or decreased cough reflex. Laryngospasm or bilateral abductor paralysis of the vocal cords may be present. Absence of tonus of the pharyngeal muscles may lead to pooling of enormous amounts of secretions in the hypopharynx. There is the usual vicious circle of increased intracranial pressure, depressed respiratory center, anoxia, cerebral edema, and hemorrhage. Tracheotomy should be performed early.

There are many other medical and surgical conditions in which tracheotomy must be considered seriously. Coma, of any origin, persisting longer than twenty-four hours requires tracheotomy. Chest injuries—such as multiple rib fractures, gunshot wounds and crushing injuries, as well as pulmonary and cardiac surgery—make breathing and coughing very difficult. Tracheotomy is a great relief in such conditions. The prognosis as to survival in extensive burns and smoke inhalation, tetanus and especially cerebral vascular accident, may be radically improved by an early tracheotomy. No patient will survive unless his airway is left open. The hemiplegic patient, especially with bulbar involvement including paralysis of the pharynx and palate, benefits greatly from an early tracheotomy.

Tracheotomy is a physiologically sound procedure. The greatly disturbed respiratory physiology becomes normal by this operation. No tissue is removed. The tube can be removed at any time within days, weeks, months, or even years, and all that is left is a little scar on the neck—a small price to pay for life saving intervention. There are no sequelae of a *correctly* performed tracheotomy. However, a high tracheotomy kept open for a long time (a technique that justifiably has been abandoned) may lead to a permanent disability.

The history of tracheotomy dates back thousands of years. Without precise knowledge of anatomy, without anesthesia, antibiotics and modern surgical technique, it was considered a most heroic (though fascinating) intervention and therefore was performed only occasionally. The soundness of its principle has always been recognized. With the development of modern techniques, it has become a safe and harmless procedure and should be performed without hesitation when needed.

Conclusions

1) Tracheotomy should be performed almost routinely in certain critically ill and comatose patients.

2) Obstruction of airways by pooled secretions accompanied by a depressed cough reflex and possibly depressed respiratory center will lead to important changes in respiratory physiology. These changes lead to anoxemia, hypercapnia and acidosis. Pulmonary edema and exhaustion of the respiratory center are further complications.

3) These grave disturbances of the normal respiratory physiology are immediately relieved by tracheotomy. The return to normal of the respiratory physiology and blood chemistry as well as the elimination of the respiratory obstruction are accompanied by a striking clinical improvement of the patient.

4) The technique of tracheotomy and its pitfalls are discussed and the importance of endotracheal intubation preceding a tracheotomy is stressed.

5) Tracheotomy is routine in polio patients in a respirator. It should be seriously considered in eclampsia of pregnancy, barbiturate poisoning, head injuries, chest injuries, extensive burns, cerebral vascular accidents, and in every case of coma lasting longer than twenty-four hours.

6) Tracheotomy is a physiologically sound procedure. Performed by a competent surgeon, it is safe and harmless.

36 Greene Street
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* * * *

POSTGRADUATE CONFERENCE IN MEDICINE

An opportunity for doctors in this area to refresh and reorient their knowledge is being offered in the form of a series of programs in postgraduate medicine, to be presented annually. The first in the series will be held January 20, 21 and 22 at the University of Maryland Hospital.

These three-day conferences are being sponsored by the Committee on Postgraduate Studies and the Medical Alumni Association of the University of Maryland. They are designed to help keep interested physicians up to date on current concepts of disease and the modern methods of diagnosis and control.

Topics will be of general interest and the daily subject matter will be demonstrated by the presentation of pertinent clinical material. Panel and group discussions are planned to provide for broader coverage of the problems encountered in clinical medicine. Featured on the programs will be guest speakers who are regarded as authorities in their respective fields.

Advance registration for the January conference should be made. A $50.00 fee, which includes banquet and luncheons, should be submitted with the application for registration. Enrollment may be made for one or two days of the program at the rate of $17.00 per day, exclusive of the banquet cost of $5.00.

This year's guest faculty includes John F. Enders, Ph.D., Chief, Research Division of Infectious Diseases, Children's Medical Center, Boston, Mass., who was a Nobel Prize Winner in Medicine in 1956; Claude J. Migeon, M.D., Professor of Pediatrics, Johns Hopkins School of Medicine; and Joseph E. Smadel, M.D., Associate Director, National Institutes of Health, Bethesda, Md.

THE VALUE OF MEPROBAMATE PREOPERATIVELY

TIMOTHY A. LAMPHIER, M.D., D.A.B.S., F.A.C.S., F.I.C.S., F.A.C.G.

An original appraisal of 184 patients studied post-operatively with Miltown* (Meprobamate) was so favorable that it was decided to evaluate the use of this drug preoperatively. A study of 56 patients was carried out, both while ambulatory prior to hospital admission and after entry to hospitals for surgery. Needless to say, the nervous and mental shock associated with the decision of a physician to carry out surgery produces tremendous fear and worry.

In addition to the so-called regular Miltown tablets, we also assayed the value of sustained action capsules of Miltown (200 milligrams) in a smaller group of 39 patients. The results of this latter study will be reported in detail at some later date.

Usually, the anxious or worried patient is not medicated prior to entry in the hospital or immediately following admission. It is frequently during this interval that too often those needing urgent surgery delay or postpone treatment until it is too late. It is fear of pain which usually frightens patients prior to surgery: e.g. prehemorrhoidectomy patients.

Frequently, and more so in the geriatric group, ambulation is desirable up to the evening before surgery. Barbiturates often will result in excessive sleep and marked lethargy.

Common complaints in the preoperative group are nervousness during the day and insomnia at night. The excellent tranquilizing effect and sleep producing qualities were apparent in 53 out of the 56 patients studied, or a failure of only 5.3 per cent. The three failures were attributable to skin reaction in one patient and gastric upset in the other two. No serious untoward reactions were observed. The patients remained alert and did not seem suppressed. Furthermore, there were no reports of addiction following cessation of the drug, or withdrawal symptoms. No euphoria or hallucinations were noted.

Total of 56 Patients Studied

Type of operation	Number of patients	Treatment days before hospitalization	Hospital days prior to surgery	Results—untoward reactions	Average age
		days	*days*		*years*
Hemorrhoidectomy	18	7	1	Excellent 0	31
Hysterectomy	12	7	2	Excellent 0	48
Subtotal gastrectomy	6	7	3	Good 2	53
Radical mastectomy	4	7	3	Excellent 0	46
Cholecystectomy	7	7	2	Excellent 0	41
Herniorrhaphy	6	7	1	Excellent 0	33
Large bowel resection	3	7	4	Excellent 0	62

Pre-operatively these patients were lackadaisical and content and did not seem to be jittery.

Surgery was purposely delayed for a minimum of one week in all cases so that the long-run effects of Miltown on this group could be fully evaluated—: prior to hospital admission.

In this study, 400 milligram tablets were administered orally—one tablet just prior to, or after each meal. There was no carryover effect and tolerance to the drug did not develop.

Furthermore, it was shown that preoperative sedation could not only be kept at a minimum but that Miltown could actually be substituted for the usual barbiturates ordered on the evening before and the morning of surgery.

Summary

1. Miltown is a valuable adjunct to preoperative preparation of the patient both before and after hospital admission.

2. Miltown can be substituted for barbiturates in routine preoperative preparation of patients for surgery.

3. Equanimity was secured without mental depression.

30 The Fenway
Boston 15, Massachusetts

* Miltown is a trade name.

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Important Reminder

ENROLLMENT IN BLUE CROSS AND BLUE SHIELD

for

MEMBERS OF THE MEDICAL AND CHIRURGICAL FACULTY

November 1958

The annual Blue Cross and Blue Shield enrollment for you and your employees occurs in November. During this enrollment period, applications will be accepted for new memberships and for changes in present coverage, including conversion to Blue Shield Plan B. In October, before the enrollment period begins, you will have received application cards and full information on the enrollment. November is the only time that Maryland Medical Service, Inc., and Maryland Hospital Service, Inc., will accept applications for insurance in Blue Cross and Blue Shield. These applications must be in their office, 200 W. Baltimore Street, Baltimore 1, by NOVEMBER 20, 1958.

TRAUMA AND CANCER[1]

SYMPOSIUM[2]

MODERATOR AND JUDGE:	THE HON. ROSZEL C. THOMSEN[3]
MEDICAL WITNESSES:	WARFIELD M. FIROR, M.D.
	W. C. HUEPER, M.D.
	WILLIAM V. LOVITT, JR., M.D.
ATTORNEY FOR PLAINTIFF:	BERNARD M. SAVAGE, ESQ.
ATTORNEY FOR DEFENDANT:	THE HON. W. ALBERT MENCHINE

PROCEEDINGS

JUDGE THOMSEN: Ladies and gentlemen, the Symposia Committee on Medical and Legal Problems of the Medical and Chirurgical Faculty, the Maryland State Bar Association and the Bar Association of Baltimore City has asked me to welcome you to this symposium on *Trauma and Cancer*.

As most of you know, the purpose of this Committee is to conduct panel discussions and have meetings on matters of common interest to the two professions. This Committee has conducted a number of very successful meetings during the past five years.

Our proceedings tonight are in the form of a mock trial. Mary Smith, the widow of John Smith, is suing the Brown Building Company (under what is quaintly known as the "Maryland Lord Campbell's Act") to recover pecuniary damages sustained by reason of the death of her husband. His death, she claims, was caused by negligence on the part of the building company, its agents, servants and employees.

For some reason, not apparent to me, the parties have agreed to try the case before a judge without a jury. I am the Judge. You must assume that the case began several hours ago, and that previous witnesses have testified to the following facts:

On June 30, 1954, Smith was a business visitor in a building owned by the defendant. As a result of an explosion, he was tumbled head over heels, a distance of about twenty feet, against a radiator. Smith was able to get up immediately but he complained of severe pain in the back and abdomen and was taken to a hospital by ambulance.

The hospital records show that he was admitted in moderate shock; blood pressure 90/50, with severe upper abdominal pains. Examination in the accident room showed marked upper abdominal rigidity with tenderness across the anterior costal margin and with contusion of the skin over the upper mid and right abdomen in an area extending from the xyphoid to the umbilicus.

The initial diagnosis was hematoma of the anterior abdominal wall and possible back injuries. X-ray of the spine failed to show any fracture. He was hospitalized for twelve days. During the first week he exhibited daily spiking temperatures to 103 degrees, generalized abdominal rigidity, vomiting for a period of three days, loss of appetite, upper abdominal and bilateral flank pain. The blood amylase on the evening after the accident was about three times the normal. By the next day it was about twice normal, and by the following day it had returned to normal limits.

He convalesced at home for another week and returned to work. During this time he suffered increasing pain in his back, which disturbed his sleep. During September and October, he noted marked loss of appetite and occasional bouts of nausea and vomiting but was up and about at

[1] Presented before the members of the Medical and Chirurgical Faculty and the State and City Bar Associations on November 20, 1957.

[2] Symposium arranged by the Joint Committee on Medico-legal Problems of the Medical and Chirurgical Faculty of the State of Maryland, the Bar Association of Baltimore City, and the Maryland State Bar Association.

[3] Chief Judge of the United States District Court.

work part of the time. In December, back pain was so severe that he stopped work. He described the pain as dull and boring, not aggravated by standing or bending but somewhat more severe when he lay on his abdomen. He took aspirin several times a day.

On February 15th, an X-ray of his lumbar and thoracic spine was reported as showing moderately severe hypertrophic arthritis with lipping of the bodies of dorsal eleven and twelve and lumbar one but no evidence of fracture or dislocation. His physician urged him to seek hospitalization but he procrastinated until April 4, 1955, when he entered a hospital.

The past history taken at the hospital at the time of his second admission showed the patient had always been well, with an average weight of 160 pounds, and no weight loss until the present illness. He had had the usual childhood diseases. He had had an episode of gastrointestinal bleeding about November 1953, which persisted for about two weeks, with two to three tarry stools a day. X-ray at that time revealed a deformity in the first part of the duodenum, but no crater. The doctor explained to the Court that this meant no deep ulceration. The patient followed an ulcer diet for about three months and had no further symptoms. Specifically he had no gastrointestinal symptomatology for the three months prior to his abdominal injury. His appetite was always good; he denied alcohol consumption. He had no respiratory or genitourinary symptoms.

A resume of the physical examination was confined to positive findings. The temperature was 100.6; pulse 100; respiration 18; weight 135 pounds. The skin was sallow. The eyes were clear. Oral hygiene was satisfactory. Heart and lungs normal. Blood pressure 110/55. There was a vague palpable mass in the upper abdomen, about three inches by two inches. There was some evidence of fluid in the abdominal cavity. In the upper third of the right abdominal muscle there was an area of fibrous thickening about one inch square. It was non-tender but the patient indicated that this was the area of his injury in June 1954. There was no discoloration at the site.

The laboratory studies were limited to significant findings. The amylase was 130 units, with slight disturbance of liver function. The gastro-intestinal X-ray series showed a mild delay in gastric emptying time, with slight dilatation of the stomach and deformity due to an extraneous mass in the region of the pancreas. The gall bladder X-rays showed no filling of the gall bladder. A flat plate suggested stones in the gall bladder area, although they were not well visualized. There were also faint "speckles" of calcium in the upper mid abdomen, suggestive of calcified fat necrosis in the head of the pancreas.

At the end of the second week he showed rapid general deterioration, and died on June 21st.

The final clinical diagnosis was carcinoma of the head of the pancreas, with abdominal metastases.

The plaintiff will call two witnesses in her attempt to prove that the death of her husband was caused by the accident, as that word "caused" is used in the statute. The defendant will call one witness. They will be examined and cross-examined by counsel: Mr. Bernard Savage[4] for Mary Smith, Plaintiff, and Mr. Albert Menchine[5] for the Brown Building Company, Defendant.

After the testimony has been concluded, members of the audience may wish to ask questions. Those questions must be written out. Ushers will collect your questions, which may be addressed to any of the doctors, or lawyers, or to me. Mr. Savage, will you call your first witness?

MR. SAVAGE: Dr. William V. Lovitt, will you take the stand, please?

Thereupon Dr. William V. Lovitt, Jr., being questioned, testified as follows:

DIRECT EXAMINATION

MR. SAVAGE: Doctor, will you give us your full name, your address, and your occupation?

DR. LOVITT: William V. Lovitt, Jr., 3501 St. Paul Street, Assistant Medical Examiner for the State of Maryland.

[4] Of the Baltimore Bar.

[5] Of the Baltimore County Bar (now Judge of the Circuit Court for Baltimore County).

MR. SAVAGE: I assume opposing counsel will concede that Dr. Lovitt is a qualified expert in this field?

MR. MENCHINE: Qualifications admitted.

MR. SAVAGE: Doctor, did you perform the autopsy in this case?

DR. LOVITT: Yes.

MR. SAVAGE: Do you have your findings with you?

DR. LOVITT: Yes sir.

MR. SAVAGE: Will you tell us what they are, please?

DR. LOVITT: The autopsy disclosed that the body was that of an emaciated, elderly, white male. It measured five feet, ten inches in length and weighed 115 pounds. There was slight jaundice of the eyes, but the skin showed no perceptible jaundice.

The body cavities were opened in the usual fashion. In opening through the abdominal wall there was found in the upper portion of the abdominal musculature an area of fibrous tissue. This showed a brownish-yellow discoloration and fibrosis typical of an old organized hemorrhage. No similar lesion is found in the left area and there were no other evidences of abdominal wall injury. The other muscles of the abdomen, both in the front and in the back, showed no evidence of injury.

Within the abdominal cavity there were approximately three quarts of cloudy, yellowish fluid. In the upper central portion of the abdomen, there was a mass of tumor which measured seven by seven by five inches. This mass was in the region of the head of the pancreas. It was not found to involve the stomach wall, but was closely associated with and adherent to the duodenum.

The surfaces of the bowel and the internal surfaces of the abdomen showed numerous small nodules of tumor. These were widely disseminated over the abdominal surfaces and were from the size of a pinhead to as much as a half-inch in diameter. These were also found over the surface of the gall bladder.

The heart showed no significant gross abnormalities.

The lungs showed only a diffuse consolidation or firmness of the right lower lobe. This was interpreted at the gross autopsy as being pneumonia. There were no tumor nodules found in the lung.

The liver was slightly enlarged. Within the left lobe of the liver there was a tumor mass. This was just above the gall bladder and measured three inches in diameter. There was no obstruction of the gall bladder or of the cystic duct. The common bile duct, as well as the hepatic ducts, were dilated and widely patent.

The head of the pancreas was replaced by a large tumor mass, seven by seven by five inches. On section, the tumor was found to be of a gray-pink color, firm, with multiple areas of degeneration necrosis. The tumor surrounded the vessels adjacent to the pancreas as well as the splenic artery and vein. There was an occlusion, or a clot, within the splenic vein. Multiple sections of this tumor also showed focal areas of calcification and fibrosis mixed with some caseous or necrotic appearing material. This obviously appeared to be fat necrosis in the pancreas and in the adjacent fatty tissues. The tail of the pancreas had an atrophic or shrunken appearance.

The spleen was slightly enlarged. There was an area of degeneration in the spleen which was hemorrhagic in color and appeared to be an area of infarction or degeneration due to occlusion of a blood vessel.

The adrenals and kidneys showed no significant gross abnormalities.

The intestines showed in the duodenal area an indented scar. This was typical of an old healed ulcer. In addition, in the midportion of the duodenum, superior to the Ampulla of Vater, was another ulcer which was half an inch in diameter. This showed a heaping up of the edges and a degenerated necrotic base. This ulcer was continuous with the previously described mass of the head of the pancreas. The remainder of the intestines showed no significant findings, other than the previously mentioned small tumor nodules which were disseminated over the surface.

The brain showed no gross abnormalities. There was no metastatic tumor.

Histological sections of the pancreas showed an extensive, poorly-organized and poorly-differentiated, glandular cancer with widespread degeneration of the tumor and many areas of hemorrhage into the tumor. The pancreatic ducts were dilated. The remainder of the organs showed no metastatic tumor other than that on the surfaces of the intestines and within the liver. All of these sections showed the same type of tumor.

A section from the area of fibrosis and old hemorrhage in the abdominal wall showed marked scarring, with many macrophages or cells filled with blood pigment. This was consistent with an area in which there was organization and repair of the area of hemorrhage.

Consequently the final diagnosis was carcinoma of the head of the pancreas with solitary metastasis in the liver and extensive nodular metastatic seeding of the peritoneal cavity over the intestines.

Question. Doctor, could you break that down into layman's language?

Answer. Well, this was a cancer of the head of the pancreas that had spread to the liver and to the surrounding tissues of the abdomen, but it had not gone to the other organs of the body.

Q. Were there any additional anatomical diagnoses?

A. Yes, lobar pneumonia with a lung abscess; a minor amount of arteriosclerotic heart disease (or hardening of the arteries) or thrombus (or a thrombosis) of the veins of the spleen with an area of degeneration or infarction of the spleen.

Q. Thank you very much, Doctor.

MR. MENCHINE: No questions.

JUDGE THOMSEN: Do you want the doctor to remain in court, or may he be excused at this time?

MR. MENCHINE: He may leave, if he wants to.

MR. SAVAGE: Yes, he may leave.

JUDGE THOMSEN: Doctor, you may leave at any time you wish.

Thereupon, Dr. Warfield M. Firor, being questioned, testified as follows:

DIRECT EXAMINATION

(By Mr. Savage)

Q. Dr. Firor, will you let us have your full name, your address, and your profession?

A. Dr. Warfield M. Firor, 1101 N. Calvert Street. I am a surgeon.

Q. Doctor, what are your qualifications?

A. I graduated from the Johns Hopkins Medical School in 1921 and spent six years going through the residencies and I have been on the teaching staff of the Medical School. Since 1929 my position has been that of Associate Professor of Surgery. I am on the visiting staff of the Johns Hopkins Hospital, the Union Memorial Hospital, and the Maryland General.

MR. SAVAGE: Any questions?

MR. MENCHINE: Yes, just one or two.

Doctor, in the last few years, have you contributed anything to the medical literature in the field of cancer?

WITNESS: Nothing.

MR. MENCHINE: I believe you have contributed to an evaluation of a treatment for arthritis within the last three years?

THE WITNESS: I would say I assisted in that.

MR. MENCHINE: I was going to ask you whether you did not have help from Dr. Lumpkin on that work?

THE WITNESS: He did the work and I signed the paper.

JUDGE THOMSEN: Have you any serious questions to ask Dr. Firor on his qualifications?

MR. MENCHINE: No, Your Honor, I don't think so.

MR. SAVAGE: I guess the doctor has been too busy helping patients to do anything in the way of writing books. Doctor, we would like to learn something about the nature of cancer. Therefore, I am going to ask you a very general question. I trust this will not be objected to, as it is only a preliminary question.

Doctor, what is cancer, what are its causes, how many types of cancer are there, what are some of the common characteristics of cancer, what is a cancerous tumor, and what is the difference between a benign tumor and a malignant one? What can be done from the standpoint of

treatment and/or surgery, if detected in the early stages, and what is the prognosis? I realize that is a broad question for an answer that may take only five minutes, but that is all the time allotted.

DR. FIROR: I accept the question and the compliment. Cancer is the lay term for malignant processes. There are several, and probably with a simple analysis, I can make it clear to the audience.

A malignant disease is an abnormal growth of cells. If one thinks of a building with the bricks on the surface all of the same size, shape and color, those bricks would correspond to the cells covering the body. Now, if suddenly a group of those bricks would change in size, shape and color, so that there were small ones and large ones, you would have the precursor to cancer. And the cells change in these characteristics. If the bricks begin to eat away and change the bricks surrounding them, that would correspond to the invasion of a malignant growth into normal tissue. Now, if the bricks would start to pull a sputnik and hop into different parts of the building and start to grow, that would correspond to metastases, or the spread of the tumor to other organs.

The difference between a carcinoma and a sarcoma is simply this: a carcinoma is a malignant growth that begins in one of the covering surfaces, like the lining of the intestines or of the skin. A sarcoma is a malignant growth that arises in one of the connective tissues.

And now we can go back to our analogy of the building. If the joists and beams that hold the walls undergo a change in shape, size and color, then you would have a malignancy of the connecting structures.

All malignant tumors arising from covering surfaces are called "carcinoma." The ones arising from connective tissue are called "sarcomas."

The second question is as to the causes of cancer. They are legion. We know of more than two hundred pure chemicals which, if injected, even in very minute amounts, into an experimental animal, will produce a malignant condition.

However, there is great specificity. The chemical which will produce cancer in a mouse will not have any effect in a rat. So there are a great number of causes of cancer.

The difference between a malignant growth and a benign growth lies in the fact that the malignant growth invades normal tissue and changes it and also jumps to other parts of the body, thereby contributing to death. Benign tumors do not have this characteristic. However, some benign tumors cause death because of the positions they occupy.

One of the commonest mistakes about cancer is that it is an entity. Nothing could be further from the truth. There are many, many more kinds of cancer than there are infectious diseases. Another common mistake is that all cancers are painful. That is not so. Still another common fallacy is that cancer is inevitably fatal. That is not so. There are some kinds of cancer that are harmless, slow growing, indolent tumors with which a person can live for thirty years. There are others that cause death in six weeks.

I would like to emphasize this one fundamental point. There are certain groups of cancers that behave and follow a general pattern, but there is nothing absolute in the field of malignancy. There are exceptions to every type of tumor. As one pathologist has said "Every tumor is its own breed of cat." And that about wraps it up in one sentence.

Q. There is one question you could touch on and that is, "What is the prognosis in cancer, if detected in the early stages, from the standpoint of treatment and/or surgery?"

A. Well, obviously, when the tumor has jumped to many parts of the body it is in the late stages and treatment is not very satisfactory. Our hope of eliminating the disease always rests upon early diagnosis and extensive removal. There are adjuvants, such as certain chemicals and irradiation, but basically the best way to eliminate a cancer is to have it diagnosed early and treated by radical surgery.

Q. Doctor, in this case the decedent is John Smith and the plaintiff is his widow, Mary

Smith. Now, when and where did you first see the patient professionally?

A. I first saw the patient at the Johns Hopkins Hospital on June 30, 1954.

Q. Doctor, I believe you treated this man a'most daily from the time of his hospital admission on June 30, 1954 (the date of the accident) until his discharge some twelve days later. You also treated him until he returned to work about July 20, 1954, is that correct?

A. Yes.

Q. Now Doctor, for the sake of brevity rather than repeat we will assume the correctness of, and follow the summary given by His Honor, Judge Thomsen, and the testimony of Dr. Lovitt as to the postmortem. Therefore, you may assume the accuracy of all the facts set forth. I assume, also, that you will consider the force of the explosion in this case which tumbled this man head over heels through the inside of a building, his body being impacted against a radiator.

Doctor, will you now summarize the significant findings on your first examination; that is, on June 30, 1954, the day of the accident?

A. The pertinent findings are as follows: The patient was in moderate shock. His blood pressure was 90/50. The pulse rate was rapid. There was severe abdominal pain. There was marked upper abdominal rigidity and tenderness and there was an evident bruise between the ribs in the upper half of the abdomen. Those were the pertinent facts on admission.

Q. I understand that you are giving us principally the significant findings and are not going into the general findings?

A. That is correct.

Q. What was your diagnosis, Doctor?

A. Well, he obviously had a contusion or bruise of the upper abdomen, with a blood clot; a hematoma was present, and possibly internal injuries.

The patient complained of back pain, but on physical examination there was nothing to indicate injury to the spine. However, that was borne in mind as a possibility.

Q. Well, Doctor, did your examination indicate that the trauma to the stomach or abdomen was severe or moderate?

A. A severe one.

MR. MENCHINE: Objected to, as to form.

JUDGE THOMSEN: How would you improve upon it?

MR. MENCHINE: I would rather have the doctor state the answer, and not the lawyer.

JUDGE THOMSEN: Objection overruled.

MR. SAVAGE: Doctor, what, if any, significance do you attach to the previous history of gastrointestinal bleeding and tarry stools in November of 1953 in so far as the condition which you found and the condition from which he died are concerned?

A. I attach no significance to it. The man was, according to his history, free from symptoms after three months of treatment for a duodenal ulcer.

Q. What treatment was given him during his first stay in the hospital?

A. He was given intravenous fluids. He was prepared for a transfusion, which he did not have. He was given sedation.

Q. Doctor, did you follow this patient from the time of his discharge from the hospital, about July 14, 1954, until the time of his second hospitalization in April of 1955?

A. Yes, because I was concerned about this patient. During his stay in the hospital, he showed evidences of internal injury.

I would like to explain to the Court that there is in the upper abdomen a large gland, called the pancreas. This is a digestive gland. It secretes material comparable to saliva, but in larger amounts. One of the functions of the pancreas is to produce a chemical, called amylase, which digests starches. It also produces a substance that digests fats. We can measure the amylase in the blood and this patient's blood showed three times the normal amount within twenty-four hours after the injury. This can be an exceedingly serious condition. So it was imperative that we follow this patient carefully. However, the amylase returned to normal in seventy-two hours.

After discharge, I saw the patient approx-

imately once every two weeks until re-admission. The story is that a few weeks after leaving the hospital he developed abdominal pain. In a few months he was losing weight, and he was re-admitted because he had lost some—I think thirty-five pounds. He was weak. He had nausea, vomiting, loss of appetite, and indigestion. All of these come with pancreatic disease.

Q. I assume, Doctor, that that was the condition for which he was hospitalized the second time?

A. That is correct.

Q. Briefly, what were your findings on the second admission?

A. There was obvious weight loss. There was no jaundice—the skin was not yellow—and I think that is significant. There was a mass in the upper abdomen in the region of the pancreas. That mass measured two by three inches. There was slight anemia. The amylase was back to normal. There was fluid in the abdominal cavity.

Q. No surgery was performed?

A. No, this patient's condition deteriorated rapidly. It seemed evident, and was proved shortly after admission, that there was fluid in the peritoneal cavity, in the abdominal cavity. Also, the size of the mass was such that surgery was inadvisable.

Q. Doctor, what, in your opinion, was the primary cause of death?

MR. MENCHINE: Objection.

JUDGE THOMSEN: I gather you just want the doctor to state the clinical cause of death without expressing an opinion as to what connection the accident had with it. Is that what you are after?

MR. SAVAGE: That is what I had in mind.

JUDGE THOMSEN: Objection overruled.

MR. MENCHINE: Objection withdrawn, then.

A. The primary cause of death was cancer of the pancreas.

MR. SAVAGE. Doctor, was there any metastasis into the abdomen, or spreading into the abdomen?

A. Yes, there was seeding all over the abdominal cavity, and tumor implants.

Q. What do you mean by "tumor implants"?

A. Bits of the tumor which are broken off and begin to grow wherever they happen to land. To make it clear I illustrated on the blackboard the pancreas, the tumor, and the multiple small tumors—the daughter tumors—the secondary growths which came from this tumor and spread all over the abdomen.

Q. Are you able to say how long this cancerous condition was present?

A. I can state it approximately. I think it was probably there for fifteen or eighteen months before death.

Q. Was this a typical cancer of the head of the pancreas?

A. No, sir.

Q. In what way was it not?

A. Cancer that begins in the right side of the pancreas, (the head of the pancreas) usually grows slowly and presses on the bile duct which brings bile from the liver to the intestinal tract. It presses on that so that bile backs up and the patient becomes yellow or jaundiced. The common garden variety of cancer of the head of the pancreas causes painless jaundice—steadily developing, slowly progressing—and when it spreads, it spreads to the liver and not all over the peritoneal cavity.

In sharp contrast, cancer of the body of the pancreas (the thin part) is a totally different disease. It begins by causing pain, unexplained pain, across the back. Shortly thereafter the abdomen fills with fluid, causing a tumor, and it seeds itself on the surfaces of the abdominal organs.

Now, I draw attention to the fact that until the last week of this patient's life there was no jaundice. I draw attention to the fact that this tumor beginning in the head of the pancreas, spread all over the peritoneal cavity, which one usually sees when the tumor is in the tail of the pancreas. So the course of this particular tumor, I think, was unusual.

Q. Doctor, can you tell whether or not the injury which the patient received on June 30, 1954 caused the condition from which he died?

MR. MENCHINE: Objection.

JUDGE THOMSEN: The question just calls for "yes" or "no." I suppose your real objection

is to the next question. Just answer it "yes" or "no," Doctor.

A. The answer is "yes."

Mr. Savage: What is your answer? And if you have an opinion, you can tell us that. What is your opinion, Doctor?

Judge Thomsen: Now, what are you trying to assume?

Mr. Savage: Well, I am asking the doctor whether or not he can tell whether the injury caused the decedent's death; and he says he can say that. And I am asking him whether or not it did cause his death.

Judge Thomsen: What are you asking him? What assumptions are you asking him to base his answer on?

Mr. Savage: I am asking him to base his answer on those assumptions which were given him in the beginning of his examination.

Judge Thomsen: Mr. Menchine, on what basis do you object:

Mr. Menchine: On that basis, I have no objection to the question.

Judge Thomsen: You can answer it, Doctor.

A. I wish to say categorically that the injury did not cause the cancer. I know of no instance in which a single trauma causes carcinoma. I do think that—

Mr. Menchine: That is objected to.

Judge Thomsen: Well, he is entitled to think.

Mr. Menchine: He has already announced that the injury did not cause the cancer, and then he is asked if the cancer caused the death. Any further answer by the doctor would be in conflict with his previous testimony and not based on anything he can testify about.

Judge Thomsen: Well, he has a right to answer the question.

Mr. Savage: Oh, well, I think I will accept the objection, because I think I can cover it in the next question. Doctor, did the trauma, or the blow that this patient received on June 30, 1954 aggravate or accelerate the cancer from which he died?

Mr. Menchine: Objected to, for the reason I gave before.

Judge Thomsen: State the basis of your objection.

Mr. Menchine: I think perhaps Mr. Savage might have some reason for it but the objection, of course, goes to the question of whether an aggravation of a pre-existing condition falls within the meaning of Lord Campbell's Act, Article 67, as a cause of death.

Judge Thomsen: Well, I think that the wording that is used in Lord Campbell's Act includes an aggravation of an existing condition which causes death, and which sooner or later would probably have occurred anyway. And with that definition of the word "caused," the doctor may answer the question.

Mr. Savage: Now, doctor, to refresh your recollection, the question is "Did the trauma which the patient received on June 30, 1954 aggravate or accelerate the cancer from which he died?

A. I think it aggravated it and accelerated it, for the following reasons: First, it is perfectly clear that the injury was directly over the tumor site. It is also clear that a rise in amylase which occurs quickly is something you see when the pancreas is damaged. It is also clear that the postmortem findings showed fibrosis and changes in the pancreas, and the inspection indicated increases which are compatible with, or which are the common result of, injury to the pancreas.

It is also clear that this was not a typical tumor of the head of the pancreas. It spread, it was scattered all over the peritoneal cavity. This one sees when the tumor is at the tail of the pancreas but which is not often seen at the head of the pancreas.

So, for those reasons, I feel that the injury caused a dissemination of the tumor and also the growth of the daughter tumors which shortened the patient's life.

Mr. Savage: Witness with you.

Cross Examination

By Mr. Menchine:

Q. Doctor, as I understand your testimony—and, I confess, I find it very difficult to understand—the reasons that compelled you to come to the conclusion you state are in combination. Is that true or false?

A. That is true.

Q. So that if any one of the premises that you accept as factual in this case is not correct, your opinion would be changed?

A. It would be slightly weakened.

Q. Let me read you something from the text of an eminent cancer specialist, Dr. Arnst.

MR. SAVAGE: Just a moment. We object. You have not indicated that Dr. Arnst is an authority, nor have you asked whether or not the doctor recognizes Dr. Arnst's authority. You can ask him that.

MR. MENCHINE: Doctor, you certainly recognize that Dr. Arnst is an authority in this field, do you not?

A. I certainly do not.

Q. You have heard of Dr. Theodore S. Malinowski, have you not?

A. Yes, I have.

Q. Do you recognize him as an authority in the field concerning blood amylase?

A. I do.

Q. Now, you have given an illustration. You have said that one of the differences between this particular cancer and what you call the typical cancer is that the cancer location in this instance is not in the location that would interfere with the—what is that little gadget that you talked about, or what was it you said there?

A. The common variety.

Q. The common variety, which this cancer did not involve.

A. If you will, it did not occlude it.

Q. Yes, on the contrary, the evidence is clear that it was not occluded at the time of death.

A. That bears me out.

Q. Would it not be significant that it was not occluded at the time of death?

A. Please repeat the question.

Q. Would it not be a significant condition that would lead to the conclusion that this injury had no connection by way of aggravation of this cancer, that the duct was open at autopsy?

A. No, I don't think so.

Q. In other words, the blood amylase you say is associated with the trauma of June 30, 1954?

A. That is right.

Q. Now, I spoke to you earlier about Dr. Malinowski. Is it not true that any mechanism that prevents a normal flow of pancreatic juice from the main pancreatic duct into the duodenum may be reflected by an elevation of serum amylase?

A. That is absolutely right.

Q. And couldn't that condition result from such causes, for example, as a mucous block?

A. Correct.

Q. And, for a cause, shall we say that a small gallstone was present only briefly in the duct and that that was removed by the milking process of elimination?

A. Quite right.

Q. And neither of those conditions would be caused or brought about by cancer, by trauma, or by injury?

A. That is right.

Q. So it is a fact, then, that blood amylase, particularly blood amylase elevation that was only for seventy-two hours could have been brought into being by a condition other than the injury which you say caused this death?

A. That is possible, but utterly improbable.

Q. Now, Doctor, you referred (as another reason for connecting this cancer with this death) to the fact that there were changes and fibrosis in the pancreas.

A. That is right.

Q. And which you said was the end result of injury to the pancreas.

A. I should have said "Could be the end result." Injury is not the sole cause of fibrosis.

Q. Might one of the causes of a condition exactly like that be a condition brought about by bleeding from an ulcer?

A. Provided the ulcer was of a type associated with an infection. Bleeding alone would not do it, but a duodenal ulcer and calcification can cause inflammation of the pancreas and that would cause the condition which you refer to as possible.

Q. At the autopsy in this case the pancreas and the duodenum were joined together, were they not?

A. They are always joined together.

Q. But by a substance of fibrosis? Isn't that the point at which the fibrosis and calcification occur?

A. No, sir, the fibrosis and calcification occurred inside the pancreas, not on the surface.

Q. And could have been produced by an ulcer, as was established at postmortem?

A. It could have been, that is true.

Q. Now, you used the expression "daughter." There is an old expression that lawyers have used—"Like father, like son." Can you say that that is true between the original cancer and the implants that come from it?

A. To a certain extent that is true.

Q. Now, you can answer this: In this case, what is the meaning of the term "original avenues of calcification"?

A. That means that a calcification of the glandular elements has occurred.

Q. That type of cancer in the region of the pancreas is a very fast-growing type of cancer, is it not?

A. No, I would not say that. Fast-growing is a relative term. To be specific, an average cancer of the pancreas runs a course of about eighteen months to two years.

Q. You mean in eighteen months to two years it reaches the point where it causes death?

A. We never know when a cancer begins. It may begin in the early stage and stay in that preliminary state for years. I would say that from the onset of symptoms until death the average duration is approximately what I said.

Q. And the daughters, so to speak, would bear a similar relationship with respect to growth?

A. Not necessarily, because sometimes the primary tumor may be no larger than a half inch and multiple tumors may be seen growing everywhere. They are the outgrowth of the parent, the primary tumor.

Q. That is what I am trying to get at; the daughters get larger than the mothers?

A. I said "Sometimes." Sometimes it happens, sometimes exactly the opposite happens. Now, the primary tumor grows very rapidly and the daughter cells very slowly. You are on very uncertain ground when trying to generalize about the behavior of secondary tumors. I have in mind a point to which usually the primary tumor has developed, but whose secondary tumors continue to grow.

Q. Will the type of cancer that behaves in the fashion you just described occur frequently in liver ducts but not in the peritoneal cavity?

A. You mean the rapidly-growing ones?

Q. Yes.

A. It comes to the liver.

Q. Doctor, you have seen in your experience, I assume, cases in which there were cancers of the pancreas without any associated indicated injury or trauma?

A. Correct.

Q. And what happened first?

A. I operated on it. I operated on the patient and excised part of the stomach, of the pancreas, and part of the liver.

Q. What happened to the patient?

A. The patient died.

Q. The second case that you had that did not involve trauma, what occurred there?

MR. SAVAGE: I do not know the significance of this question because there was no surgery performed in this case. Therefore, I object to the question.

JUDGE THOMSEN: Do you contend that the presence or absence of surgery had anything to do with it, Mr. Menchine?

MR. MENCHINE: No, I do not maintain that, but I maintain that there is a cause for death in this case not connected with cancer, or with injury, and I want to bring that out by cross-examination.

MR. SAVAGE: You cannot do it by proving what you say you are trying to prove.

JUDGE THOMSEN: I think he can answer the question.

MR. MENCHINE: I tried to prove it, Mr. Savage, by proof of death.

JUDGE THOMSEN: It is part of the cross-examination. The doctor may answer the question.

Q. (By Mr. Menchine) Will you tell me about your second and third cases, Dr. Firor?

A. In both cases, the patient died.

MR. MENCHINE: No further questions.

REDIRECT EXAMINATION

MR. SAVAGE: Doctor, how about the fourth case?

A. She is still living after six and a half years.

Q. That is an actual case, isn't it, Doctor?

A. That is correct.

Q. Doctor, we were not dealing with death by cancer of the pancreas, were we?

A. We saw the condition that existed there.

Q. You take great pains, so far as you can, to find out the cause of cancer of the pancreas?

A. Yes.

MR. SAVAGE: That is all.

JUDGE THOMSEN: Doctor, there are several questions I would like to ask you. Mr. Menchine asked you for a further explanation of amylase, which you testified might possibly have been caused by a mucous block or by a small gallstone temporarily in the duct. Did you find any evidence of mucous block?

THE WITNESS: No, and there would not have been detectable evidence without operation.

MR. MENCHINE: Thank you for your fairness, Doctor.

JUDGE THOMSEN: Do you think this condition might just as likely have been caused by a mucous block as it was by the injury?

THE WITNESS: No, I don't think you need to invoke the passage of a gallstone or the presence of a mucous block when you have an injury as severe as this. The injury was quite capable of causing changes in the pancreas and in the amylase.

JUDGE THOMSEN: There were some questions about whether the calcification in the pancreas could have been caused by the ulcer which this man had had shortly before the accident. I believe you said it could have been so caused. In your opinion, was it so caused?

THE WITNESS: I think it highly improbable because the patient's symptoms at the time he had the duodenal ulcer were not those of pancreatic involvement. When an ulcer appears to be deeply seated, is inflamed and, in fact, causes a secondary pancreatitis, there are signs and symptoms which will indicate it. When we treated this patient first, for bleeding, there was no evidence of pancreatitis.

MR. SAVAGE: I have just one more question. Doctor, in reference to the blood amylase immediately upon admission, what was the count? Was it normal, and if not when did it come back to normal?

A. The amylase values normally run about 150 units for 100 cc. of blood. After admission it was 450, a threefold increase. The next day it was 250. The following day it was back to normal.

Q. Which would indicate, as I believe you said, a severe blow to the pancreas itself, or a disturbance of the pancreas?

A. Yes, it would indicate that.

MR. SAVAGE: Thank you, Doctor.

JUDGE THOMSEN: Any other questions?

MR. MENCHINE: No further questions, Your Honor.

(Statement concluded)

MR. MENCHINE: Dr. Hueper, will you take the stand, please.

Thereupon, Dr. W. C. Hueper, being questioned, testified as follows:

DIRECT EXAMINATION

By Mr. Menchine:

Q. Doctor, what is your name and present occupation?

A. I am Dr. W. C. Hueper, Chief of the Environmental Section of the National Cancer Institute, Bethesda, Maryland. I am also a consultant on employee's compensation with the United States Department of Labor.

Q. Doctor, what training have you had in the cancer field?

MR. SAVAGE: I will be very happy to admit the Doctor's qualifications.

MR. MENCHINE: Just for the information of the Court, Mr. Savage, I would like to go very briefly into the Doctor's general training and experience in this particular field.

JUDGE THOMSEN: You may go into it.

MR. MENCHINE: Doctor, you have contributed to the medical literature in the field of cancer, have you not?

A. Yes, I have written some articles for publications and have made several contributions to books. I wrote a textbook on "Occupational Cancer and Allied Diseases." I am a diplomate of the Board of Pathology and of the Board of Preventive Medicine.

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...in Rheumatoid Arthritis: Impressive therapeutic effect in most cases of a group of 89 patients[4]... 6 mg. of ARISTOCORT corresponded in effect to 10 mg. of prednisone daily (in addition, gastric ulcer which developed during prednisone therapy in 2 cases disappeared during ARISTOCORT therapy).[5]

1. Rein, C. R., Fleischmajer, R., and Rosenthal, A. L.: J. A. M. A. *165*:1821, (Dec 7) 1957.
2. Shelley, W. B., and Pillsbury, D. M.: Personal Communication.
3. Sherwood, A., and Cooke, R. A.: Personal Communication.
4. Freyberg, R. H., Berntsen, C. A., and Hellman, L.: Paper presented at International Congress on Rheumatic Diseases, Toronto, June 25, 1957.
5. Hartung, E. F.: Personal Communication.
6. Schwartz, E.: Personal Communication.
7. Sherwood, A., and Cooke, R. A.: J. Allergy 28:97, 1957.
8. Hellman, L., Zumoff, B., Kretshmer, N., and Kramer, B.: Paper presented at Nephrosis Conference, Bethesda, Md., Oct. 26, 1957.
9. Ibid.: Personal Communication.
10. Barach, A. L.: Personal Communication.
11. Segal, M. S.: Personal Communication.
12. Cooke, R. A.: Personal Communication.
13. Dubois, E. L.: Personal Communication.

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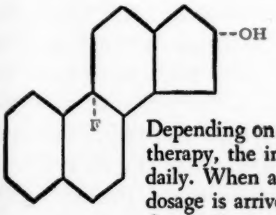

Depending on the acuteness and severity of the disease under therapy, the initial dosage of ARISTOCORT is usually from 8 to 20 mg. daily. When acute manifestations have subsided, maintenance dosage is arrived at gradually, usually by reducing the total daily dosage 2 mg. every 3 days until the smallest dosage has been reached which will suppress symptoms.

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MR. MENCHINE: All right, Doctor, thank you. Your witness for cross-examination, Mr. Savage.

JUDGE THOMSEN: He admitted his qualifications. Have you any questions?

MR. SAVAGE: No, I don't think so, Judge.

MR. MENCHINE: Dr. Hueper, have you heard all of the evidence in this case?

A. Yes, I did.

Q. As a result of hearing all of the testimony and assuming the truth thereof, with the exception of the testimony of the expert witness on the side of the plaintiff, are you able to express an opinion, with reasonable medical certainty, as to whether there was or was not a causal connection between the accident that has been described in the testimony and the death of this poor, unfortunate man?

A. I think the evidence at hand shows quite clearly that the trauma was not the primary cause of the pancreatic cancer.

Q. What are your reasons for that conclusion, Doctor?

A. There is no scientific evidence in medical literature (at least no competent evidence), which would say a single acute trauma to normal tissue could produce a cancer.

MR. SAVAGE: I object to the question and I move that the answer be stricken out because it is beside the point. We do not say that the injury caused it. We say it was aggravated or accelerated by the injury.

MR. MENCHINE: In view of the fact that he asked the doctor that same question, I thought it was all right for me to ask it.

JUDGE THOMSEN: I think that counsel will agree that if we can go on that assumption, Mr. Menchine will ask another question.

MR. MENCHINE: I am asking for an opinion, expressed with reasonable medical certainty, as to whether there was association by way of aggravation between the trauma or the injury that has been described in the evidence and this man's death.

MR. SAVAGE: If you add "acceleration," I won't object to your question

MR. MENCHINE: I will accept the amendment.

THE WITNESS: I do not believe that the evidence indicates that the trauma had affected the pancreas at all or that it accelerated the pre-existing cancer of the pancreas.

MR. MENCHINE: Why do you say that, Dr. Hueper?

A. The fact that the man had an increased amylase content of the blood for about two or three days only indicates that there was not a severe trauma to the pancreas. It was a very transitory episode which could not have produced any severe anatomical changes in the pancreas.

Q. Doctor, would you explain the statement in the light of the postmortem proof with respect to the fibrosis and calcification?

A. If a severe trauma to the pancreas had been present, we would have to expect that there would have been adhesions of the omentum with the traumatized part of the pancreas which is ordinarily found if severe trauma is exerted on any part of the abdominal cavity.

Q. Does the postmortem rule out the possibility of such adhesions?

A. There were no adhesions, according to the postmortem report.

Q. Did the postmortem report say that there were none?

A. Probably not. It was not mentioned in the postmortem report. Secondly, the histological examination of the pancreas did not reveal the presence of any old blood pigment. If there had been a severe trauma to the pancreas, we should expect that a hemorrhage was there, similar to the hemorrhage in the abdominal wall, as a result of an old hemorrhage. Blood pigment histologically was not seen. That, again, is a piece of evidence which does not support the claim that there was any severe trauma with an anatomical injury to the pancreas.

Q. Dr. Firor, in his testimony, mentioned the elevation of the blood amylase. In your opinion, is the fact that there was such an elevation on June 30, 1954—and that the elevation of the amylase continued for seventy-two hours—would that have any significance for you on the question of the aggravation or acceleration with respect to the injury?

A. I do not believe that even if a slight trauma to the pancreas occurred, it could have had any significant effect on the course of a pre-existing cancer. Certainly, however, a severe trauma to a peritoneal cancer might accelerate the growth, and if the trauma was severe, we could expect it in this case. We do not get a significant aggravation from biopsy procedures. I am quite sure that if we did, then surgeons would avoid doing them.

Q. Is there any evidence that the biopsy caused a metastasis of the cancer?

A. No, if it would cause metastasis of the cancer, they would surely avoid using such diagnostic measures.

Q. What, if any, significance is there in the finding of implants in the peritoneal cavity?

A. According to the histological data in the postmortem report, the cancer had a structure which was quite immature. That is, it had a structure which would indicate that it was a rather fast growing cancer—what we call an anaplastic cancer. It was not the garden variety of cancer, as was pointed out. It was an unusual type of cancer. It was for this reason that the cancer behaved in one respect a little bit differently from the usual type of cancer of the head of the pancreas. It not only metastasized, but it spread into the liver and also produced smaller metastases in the peritoneal covering, or the peritoneum. However, the metastasis in the liver was very much larger than the metastases in the peritoneal wall. That means that the metastasis in the liver was produced very much earlier than the metastases in the peritoneum. Therefore, it is more than unlikely that these metastases—the abdominal metastases—were produced as a result of the trauma.

Q. Why do you say that, Doctor?

A. The difference in size between the metastasis in the liver and the multiple metastases in the abdominal wall would indicate that they originated at different times. We would expect that peritoneal metastases produced by a rapidly growing tumor would develop within the ten or twelve months—which elapsed between the trauma and the death of the patient—into nodes of a very much larger size than those described in the postmortem report.

Q. Now, are there any other reasons that compel you to the conclusion which you have stated?

A. Yes, there are other reasons. The history of the patient indicates that he had a duodenal ulcer. The postmortem report indicated that the duodenum was adherent to the pancreas. There were inflammatory changes and adhesions between these two organs. The man had a hemorrhage from this old duodenal ulcer. He had an ulceration that was deep and affected one of the larger vessels in the duodenal wall. Extension of inflammation into the pancreas under such circumstances is most likely. For this reason, I believe that the fibrosis of the pancreas was not the result of the trauma but developed long before the trauma as a result of the duodenal ulcer.

Q. Any other reasons, Dr. Hueper, in connection with your testimony?

A. Yes, I think that there is another reason. The significance of fat necrosis and the fat calcifications was minimized. The patient was brought into the hospital after the injury in a state of shock. This suggests that he consumed no food for the first day or two. Fat necrosis and subsequent calcification of the pancreatic fat tissue will occur only if pancreatic juice is released from the traumatized pancreas into the free abdominal cavity, or into the surrounding pancreatic tissue. This occurs only under the stimulus of ingested food. Since the patient evidently did not ingest food under the condition of shock, there is no reason to believe that any pancreatic juice was released. So, I say again that the previous ulceration of the duodenum resulted in the liberation of the pancreatic juice once the pancreas was inflamed. Therefore, the occurrence of the calcifications had no connection with the trauma to the pancreas.

Q. The incident of abdominal bleeding by the ulcer was how long before the injury, Dr. Hueper?

A. I think about six or ten months before.

Q. Is there any significance in the elevated

blood amylase in a period of forty-eight to seventy-two hours?

A. I think I have already answered that question.

Q. Well, I was uncertain on that point; if a trauma had brought about an elevation of blood amylase, would it be expected to be of longer duration than seventy-two hours?

A. One would expect prolonged increase of the blood amylase if there was a severe or a significant injury to the pancreatic tissue, if the trauma produced destruction of the pancreatic tissue.

MR. MENCHINE: Your witness, Mr. Savage.

JUDGE THOMSEN: Before the cross examination starts, the ushers will pick up the questions that you would like to propound to the members of the Panel. If you have not yet framed your questions the ushers will pick them up after the cross examination.

Now, Mr. Savage, you may cross examine the witness.

CROSS EXAMINATION

Q. Dr. Hueper, your time is spent principally with pathology and experimentation with mice and rats and writing articles, rather than in the treatment of patients, is that correct?

A. I did some medical practice, too.

Q. Your experience in surgery on human beings is not very extensive, is it?

A. No, it is not.

Q. Doctor, in this case we are dealing with a very severe trauma to a man's organs.

A. I will concede that there was considerable trauma to his abdominal wall.

Q. So much so that evidence of it was found at the postmortem a year after the original injury, is that correct?

A. That is right.

Q. Of course, trauma of the pancreas is a relative term, and it can be severe or it can be slight. Is that true?

A. Mr. Savage, trauma is a multiple daily occurrence throughout life.

Q. Yes, but in this case the trauma was apparently severe enough to produce an increase in the blood amylase of three times the usual rate.

A. That is right.

Q. So there was definite evidence of trauma to the pancreas, itself; is that correct; about that there can be no doubt?

A. No.

Q. It may not seem significant to you, but there was evidence of trauma; is that correct?

A. No, that is not correct. If you understand it, we assume here that the trauma caused an anatomical injury to the pancreas. There may have been a temporary functional disturbance of the pancreas which may have been produced by the shock condition. That I will admit.

Q. All right, whether you call it shock or whether you call it trauma, the effect is the same from an external force. Is that correct?

A. No, the effect is not the same.

Q. But it is one produced by external force; is that correct?

A. Oh yes, it was produced by an external force.

Q. Doctor, I believe you said that the increase or the rate of growth of the cancer in the liver was much greater than that in the pancreas. Did you say that?

A. That is right.

Q. You attach some significance to that, do you not?

A. Yes, to a certain extent, yes.

Q. Well, does the blood supply have anything to do with the growth of a cancer?

A. It has.

Q. As a matter of fact, isn't the blood supply in the liver much greater than the blood supply in the pancreas?

A. You cannot count it that way. In an abdominal cavity the cancer cells are bathed in a nutritional fluid. It is just like tissue culture growth.

Q. But you do admit that the blood supply in the liver is three times greater than in the peritoneum?

A. In the peritoneum, yes.

Q. All right, we agree on that. Now, you said that in this case it was indicated that there was no blood pigment at the autopsy. I suppose you

mean by that, evidence of hemorrhage. For instance, if you strike your arm and cause a bruise, the bruise is the blood pigment, isn't it?

A. No, the blood which exudes from a torn vessel would be decomposed and would form blood pigment.

Q. Yes, and after a time it would pass off into the system and there would be little or no evidence of it. I think that is possible.

A. I think Mr. Savage is not an eminent pathologist.

Q. Well, Doctor, it is perfectly possible to have a trauma without having blood pigment; isn't that true?

A. I guess we are again discussing what is trauma to the pancreas in this case?

Q. Yes.

A. If you have a traumatized pancreas, you would be bound to have a hemorrhage. If you have a hemorrhage you should have in the traumatized fibrous tissue which is formed old blood pigment, and that blood pigment provides the pathognomonic symptoms and characteristics of an old hemorrhage.

Q. There was blood pigment in the abdominal wall, wasn't there?

A. Not according to the postmortem report.

Q. Well, there was definite evidence of severe trauma to the abdominal wall a year afterwards.

A. That was within the abdominal wall.

JUDGE THOMSEN: The memorandum I have of what Dr. Lovitt said is that the abdominal wall section showed scarring with many macrophages in the fibrous tissue, or scar tissue, and that this section is very hematomic in the upper abdominal area.

THE WITNESS: That is a typical description of an old hemorrhage, only in this case the site of this old hemorrhage is the abdominal wall and not the pancreas. It is a fundamental difference.

MR. SAVAGE: So you will admit that there was a severe trauma to the abdominal wall?

A. Oh, yes, I totally agree to that.

JUDGE THOMSEN: Now, wasn't there some fibrosis between the pancreas and the duodenum?

THE WITNESS: Yes, there was.

JUDGE THOMSEN: What light does the report of Dr. Lovitt shed with respect to that fibrosis, as to whether or not it contained this pigment?

THE WITNESS: It was a duodenal ulcer which bled into the duodenal lumen and into the tissues.

MR. SAVAGE: And that the fibrosis was at the site of the old ulcer.

A. That was beneath the old ulcer.

Q. Yes. Now, Doctor, it is your finding that there was a hemorrhage some nine months before this injury. Is there evidence in this case of pancreatitis at the time of the hemorrhage?

A. You mean symptomatically?

Q. Yes.

A. Not according to the record, but according to the histological statement which I interpreted.

Q. According to the records, there was no evidence?

A. No.

JUDGE THOMSEN: What do you mean by "histological," Doctor? I am not quite sure that I understand it.

THE WITNESS: Histological changes which were found at microscopic examination in this particular pancreas, in that particular region of the pancreas.

MR. SAVAGE: Doctor, if it is your thought that this blood ulcer caused changes in the pancreas, why weren't there symptoms during that time?

A. There do not need to be. If the pancreas was not involved, was not obstructed, there is no reason why there should be any marked symptoms.

Q. There would be some?

A. He surely had symptoms when he had the duodenal ulcer, there is no doubt about it, and he probably had very severe symptoms.

Q. Not according to the history here, Doctor. We have to go on the history we have here. We cannot assume. We must stick to the evidence. Doctor, do you recognize this statement about secondary traumatic cancer? "The adoption of this viewpoint" (that is about the absence of causes of traumatic cancer), "however, does not completely eliminate traumatic factors as significant contributory influences in the causation and precipitation of the development of cancers

or in their aggravation under special circumstances."

MR. MENCHINE: I object to Mr. Savage's use of "causation," the quotation from an expert here, unless it is acknowledged by the witness here that he is an expert in the field.

MR. SAVAGE: I think that objection is well taken. Doctor, I assume you recognize yourself as an expert.

A. Well, I think any medical man who appears in court for testimony is rated as an expert.

Q. Thank you Doctor; that was your own article.

A. I recognized it.

Q. It was published in 1955, in the American Journal of Pathology. That is still your opinion, is it not?

A. That is correct.

Q. You also stated in that same article "The traumatic tissue reactions seem to act in the form of a trigger mechanism upon a tissue specifically prepared and presumably possessing precancerous or latent cancerous alterations." That is still your opinion, is it not?

A. That is right.

MR. SAVAGE: Thank you very much, Doctor.

REDIRECT EXAMINATION

By Mr. Menchine:

Q. Doctor, just one question. You spoke about the implants that were found in the peritoneum. You described them as having a situs in what I believe you called the culture medium. What do you mean by that?

A. The abdominal wall, or the peritoneal wall is bathed in a considerable amount of serous fluid which acts as a culture medium for any growth which may become attached to it.

Q. Does that aid in the growth of those implants?

A. Definitely, yes.

Q. Would you expect implants of this size to grow as a result of a trauma that happened thirteen months before?

A. Not of that size.

MR. MENCHINE: That is all.

RECROSS EXAMINATION

MR. SAVAGE: Of course, Doctor, the growth rate of the implant is variable, isn't it?

A. That depends somewhat upon the histological type of a tumor.

Q. That is right. I mean, it can grow slow or fast?

A. Yes.

Q. Or, as Mr. Menchine very aptly put it, the daughters are sometimes bigger than the parents, and sometimes the reverse; is that true? I mean that there is no set pattern?

A. You mean following accidents?

Q. All right, that is another way of saying it.

A. You see, there is a difference whether we make it a rule or whether we make it an exception. I would make it the exception.

Q. I see; and I believe you heard Dr. Firor's testimony that this cancer of the pancreas was not of the usual type?

A. Oh yes, that is a different type. That is not the usual type.

Q. You agree with that?

A. Yes, it was a rapidly growing cancer.

MR. SAVAGE: All right, Doctor, thank you very much.

JUDGE THOMSEN: Just one final word before the question period. As you have probably guessed, these facts were taken from a real case. However, they were so altered as to leave considerable room for a bona fide difference of opinion as to causation. Furthermore, I think it is only fair to say that counsel have made more objections than they would normally have made in a case in order to give some opportunity for rulings; to clarify some matters that would not be obvious to many people here. Finally, somebody asked me whether I was going to make a ruling on the case. Well, I am not going to do so because I am sure it would give away a secret that I have been trying to hide for five years, that is, whether I am a plaintiff's or a defendant's judge. So just kind of relax for a couple of minutes until we can see if there are any more questions you want to give to the doctors here.

There is one question that I think we should ask now. It has been suggested that it may not

be inappropriate to see which group—the legal or the medical—is more interested in this type of meeting. Therefore, to see how many doctors are here, as compared to the number of lawyers—will the doctors in the room stand up?

(Members of the audience then stood up).

MR. SAVAGE: We estimate that there are about seventy-five doctors.

JUDGE THOMSEN: And how many lawyers?

(Members of the audience then stood up).

JUDGE THOMSEN: It is about two to one.

MR. SAVAGE: I would say about one hundred and fifty lawyers.

JUDGE THOMSEN: (Reading question passed up by member of audience) The first question is for Dr. Firor.

Q. Dr. Firor, how long do you think the deceased would have lived if there had been no trauma?

DR. FIROR: Is this off the record?

JUDGE THOMSEN: The case is over.

DR. FIROR: I really do not think the trauma had anything to do with it. He would not have lived ten minutes longer.

JUDGE THOMSEN: The next question: Isn't it true that some experts feel that biopsy does increase the rate of speed of metastasis and that, therefore, the blow could have caused a biopsy trauma?

DR. FIROR: "Biopsy" means when you cut into a tumor to take a piece out for diagnosis. Some experts do feel that where you cut into a very bloody tumor, it increases the likelihood of the breaking off of cells into the bloodstream. Therefore, we make it a rule when biopsying a tumor to be ready to do the big operation immediately rather than to allow a lapse of several days between the biopsy and the definitive surgery.

JUDGE THOMSEN: The next question is for Dr. Hueper. It is: Do you know of any instance where a single trauma has resulted in a cancer in any part of the body?

DR. HEUPER: No.

JUDGE THOMSEN: And another one: Are we to understand that trauma to a cancer does not cause spread in some cases?

DR. HUEPER: I concede traumatic aggravation only if there is definite evidence of an acceleration of what we call the normal course of the disease, and that the tumor spreads either from its original focus more rapidly right after the trauma, and that we have a sort of explosive growth of the tumor from the traumatized tissues. Under these conditions I would acknowledge that the trauma had something to do with the acceleration of the course of the disease.

I would recognize aggravation if one observes soon after the trauma a rapid spread of metastases. However, the darkly pigmented tumors (the skin melanomas) will produce showers of metastases at times without any trauma whatsoever.

I would concede that if a biopsy taken from an intraosseous sarcoma, which is still encapsulated by bony tissue, is followed by a rapid spread of the tumor and metastases, that any metastases which occurred after such an event might be caused by the biopsy. This has been observed. We have never biopsied melanomas. If one did, one might see a very rapid spread of the tumor after such surgical trauma. There are some exceptions to the innocuousness of trauma to a cancer from biopsy, but as a rule biopsy trauma does not act on the growth rate or cause acceleration of cancer.

JUDGE THOMSEN: Mr. Waters has a question.

MR. WATERS: If Your Honor pleases, I would like to ask Dr. Firor whether or not cancer cells exist in all human beings.

DR. FIROR: I have not examined all human beings, but I accept the question without the implication. If by a cancer cell, you mean a cell which has been altered in size and shape and is potentially capable of going on and becoming malignant, then I think we all have such cells in us. I am sure I have fifteen or twenty embryonic cancers in me right now. However, most human beings have the mechanism for keeping these incipient cancers under control.

There is evidence to show that in the uterine cervix there may be changes which may be cancerous when examined under the microscope but which we know have been there fifteen years

before their expansion and spread begins. So that preliminary changes probably occur many times in the life of the normal, healthy person. This points up our complete ignorance of the factors which are necessary for invasion and metastases.

How is it that a cancerous area will stay dormant? Sometimes we see a cancer, and we take it out, and the patient lives ten years, and then suddenly blossoms out with metastases everywhere? Those secondary growths must have occurred before the primary tumor was removed but they can remain dormant for ten or fifteen years.

I had one patient and thirty-one years after a cancer had been removed she turned up with a cancerous growth in the scar. Now, that is pretty good evidence that the body has a mechanism for keeping malignant growth under control.

One might spend hours talking about experimental evidence of resistance to cancer. And if by "cancer" you mean altered cells that evidently have the ability to invade and erode other parts of the body, then I think everybody here has some of those tucked away in one of their organs. However, probably all of you have adequate capacity for keeping them there. Does that answer the question?

MR. WATERS: Very well done, Doctor.

JUDGE THOMSEN: Dr. Hueper, in what manner, other than by biopsy, can cancer be ascertained to be present, with any degree of certainty, at a stage early enough to be treated and/or checked?

DR. HUEPER: I do not think there can be any question that the histological diagnosis of cancer is the most reliable one. It is not one hundred percent reliable, but it is by far the best one.

In recent years the so-called cytological smear diagnosis has been introduced. It is quite helpful as a screening method. However, I do not think anyone would remove a lung from my chest only on the basis of a cytological smear made from my sputum. I do not think diagnosis by that method is good enough to justify major surgery. If the diagnosis is positive, however, I think further examination should be made to determine whether or not cancer is present. X-ray diagnoses are even less reliable than the histological diagnosis or the cytological diagnosis. As a rule we will be able to demonstrate cancer by x-ray examination at a much later stage than with the histological diagnosis.

JUDGE THOMSEN: Mr. Menchine, you are asked one thing; that is "What did Dr. Malikowski really say?"

MR. MENCHINE: I do not have the book, but what I read is really about what he said, unless I was a little too busy to write it down exactly as it was.

JUDGE THOMSEN: Dr. Firor, have you ever told your patients prior to operation, that the operation would speed up that cancer? If so, then did the patient accept your operation?

DR. FIROR: No. We have many patients from whom we extirpate the cancer completely. We have not felt that in any manner this is going to accelerate the growth, and it is not necessary to tell people that an operation might do so.

JUDGE THOMSEN: Here is a question: "Could an exploratory laparotomy have been performed in the early stage (before three quarts of fluid formed in the peritoneum), etc. Were you not waiting for a textbook case before making a final diagnosis"?

DR. FIROR: Never having seen the patient until tonight, I do not think I would have. The point is, could an abdominal operation have been done earlier to establish the diagnosis. Now, one cannot answer that question without having actually seen the patient. We do not have enough factors and data about this man to say that at the second admission he should have had an abdominal operation. That is not given us.

JUDGE THOMSEN: Well, ladies and gentlemen, these meetings are scheduled to last until ten o'clock. It is now two minutes of ten, so the meeting is adjourned.

BLUE CROSS • BLUE SHIELD

Medical and Chirurgical Faculty members (including their families and employees) may enroll from November 1 to November 20, 1958 in Maryland Medical Service, Inc., and Maryland Hospital Service, Inc.

YOUR MEDICAL FACULTY AT WORK

JOHN SARGEANT, *Executive Secretary*

COUNCIL

The Council of the Medical and Chirurgical Faculty met at the Commander Hotel, Ocean City, Maryland, on September 11, 1958, and took the following action:

1. Approved a revised fee schedule and policy for use of facilities at the Faculty Building.

2. Endorsed action of the Executive Committee in specifying that it, "disapproves the use of Hypnosis by other than someone exceptionally well-trained in the field of hypnosis and qualified with the degree of Doctor of Medicine."

3. Authorized the Chairman of the Council to designate a representative of the Faculty to attend the February 16, 1959, meeting of the A.M.A. Industrial Health Congress in Cincinnati, and pay his expenses.

4. Approved the present procedures for handling:

   (a) Queries from physicians for locations in Maryland

   (b) Queries from communities needing assistance in attracting a physician to their community.

   (Physicians requesting locations are provided an up-to-date booklet listing areas in Maryland needing physicians, together with data about the community involved.

   (When queries are received from communities, the local component medical society is contacted to obtain permission for listing such a community as being in need of a physician. Once this approval is obtained, information is requested from the community from which the data sheets referred to above are prepared.)

5. Authorized publication in the Maryland State Medical Journal of lists of newly licensed physicians in Maryland and lists of license revocations, suspensions, etc., as they occur.

6. Established the date for the 1959 Semiannual Meeting as September 18, 1959.

7. Authorized retention of services of Mr. G. C. A. Anderson for the purpose of defending physicians.

8. Heard the following report and discharged the committee assigned to this problem:
   Report of Committee Regarding Choice of Physician Under Workmen's Compensation Law.

HOUSE OF DELEGATES

The House of Delegates at the Semiannual Meeting held on Friday, September 12, 1958, in Ocean City, Maryland, took the following action:

1. Approved miscellaneous By-law changes; and accepted certain Constitutional amendments for action at the Annual Meeting in April.

2. Heard various committee reports as follows:

   (a) Report from Class A Members of the Maryland Medical Service, Inc., regarding the April, 1958, resolution which requested an investigation as to the feasibility of transferring certain services from Blue Cross to Blue Shield.

   (b) Endorsed the Liaison Committee on Accreditation of Hospitals Report, and authorized this Committee to transmit to the Council on Medical Education and Hospitals of the A.M.A., certain complaints received by the Committee and to make two specific suggestions for improvement of the national committee's work.

(c) Report of the Negotiator for the Medicare Fee Schedule and Report of the Medical Advisory Committee for the Medicare Program were received by the House. This dealt chiefly with the recently published medicare fee schedule, as well as the operation of the program in Maryland for the fiscal year just ended. Information report only.

(d) New Building Committee Report advised that around $153,000 now was invested in the Building Fund; it was drawing interest; and that concrete suggestions would be brought forward by this Committee for action at the annual meeting.

(e) Committee to Investigate Problem of Practicing Physicians in Government Service reported that such physicians were not allowed to practice in any community without approval of the local commanding officer and local medical officials. The Committee was discharged with thanks.

(f) Committee to Investigate Group Insurance on a state-wide basis reported progress in investigating various types of policy with the advice that more concrete information would be available by the time of the Annual Meeting.

(g) Report of the delegate to the A.M.A., which reported on the activities at the June A.M.A. meeting and also requested an invitation be extended to the local Student A.M.A. to appear at the next House of Delegates meeting. This was approved.

COPIES OF ALL REPORTS ARE AVAILABLE FROM THE FACULTY OFFICE AT THE REQUEST OF ANY MEMBER.

The House of Delegates also passed several resolutions, which are summarized as follows:

1. Accreditation of Hospitals and Intern and Resident Training Programs.

This resolution deals with the present methods of accreditation by the Joint Commission on Accreditation of Hospitals and also with approval by the A.M.A. Committee dealing with Resident and Intern Training Programs. It urges a reevaluation of the present methods and suggests several changes in the manner in which surveys are conducted by the Joint Accreditation teams. Introduced through the Baltimore City Medical Society, by Dr. Samuel Morrison.

2. Health & Welfare Committees of the Senate and House of the General Assembly of Maryland.

This urges the formation of a Health and Welfare Committee as outlined above. This committee would deal with all health and welfare matters introduced in the Maryland Assembly and report back to the various Houses on approval or disapproval of these legislative measures. Introduced by Montgomery County Medical Society.

3. Laboratory Facilities of the State Department of Health.

This dealt with use of laboratory facilities of the State Department of Health by patients able to pay for such services, except where no private laboratory facilities existed.

The resolution, introduced by the Maryland Society of Pathologists, calls for better education of the practicing physician in not referring such patients to State Health Department facilities; and also urges the State Department of Health to take steps to curtail such service to patients able to pay for same.

4. Inequities in Blue Cross/Blue Shield payments to Washington County Physicians.

Introduced by Washington County Medical Society, the resolution urges the Blue Cross/Blue Shield Officials to evaluate the fees and manner in which they are paid for surgical assistance at operations; electrocardiograms; and for emergency room work.

5. Extension of Services under Blue Cross Coverage.

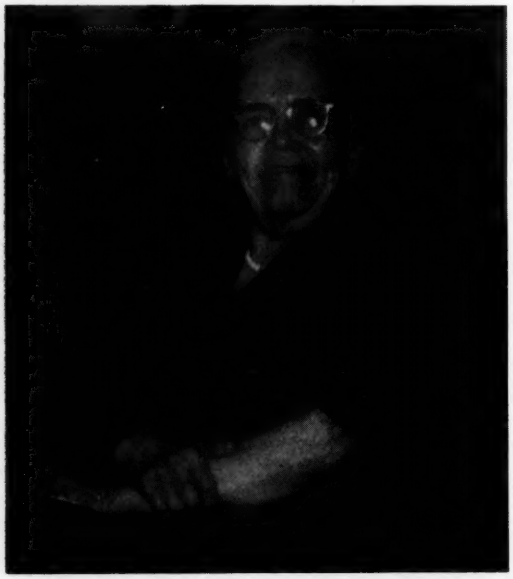
This resolution was referred to the House by the Council and provides that the Faculty express its opposition to extension of diagnostic medical services in out-patient clinics of hospitals under Blue Cross.

The House also approved the listing of a voluntary contribution for the American Medical Education Foundation on dues bills to be sent out in the future. The amount of such a voluntary contribution was established as $10.00.

Component Medical Societies

ALLEGANY-GARRETT COUNTY MEDICAL SOCIETY

LESLIE E. DAUGHERTY, M.D.
Journal Representative



The late Dr. Charlotte Gardner, who was the first pathologist in Allegany county.

Dr. Gardner, who died on August 18th of this year, was one of three women physicians ever to have practiced in Cumberland and the first of the six to practice in Allegany county. She and Dr. Catherine H. Buell were the first women physicians to locate there.

Other women physicians in the County are: Dr. Hilda J. Walters, Frostburg; Dr. Elizabeth Brings, Cumberland; Dr. Mildred Sheesley Wagner, Westernport; and Dr. Ruth Peachy, Grantsville.

Dr. Gardner was Allegany county's first pathologist and had been in practice since 1905.

LONACONING MEDICINE

The first physician to locate in Lonaconing was a Dr. Hunt, followed by Dr. Wundruh and succeeded by Dr. Herman, who came from Baltimore.

Lonaconing as a village had no existence until 1837. It lies in a valley between Frostburg and Westernport. It was incorporated in 1890, about fifty years after its founding.

In the year 1837 mining of iron was commercially introduced, and the Company doctor came into being. His fee was fifty cents per able bodied man per month.

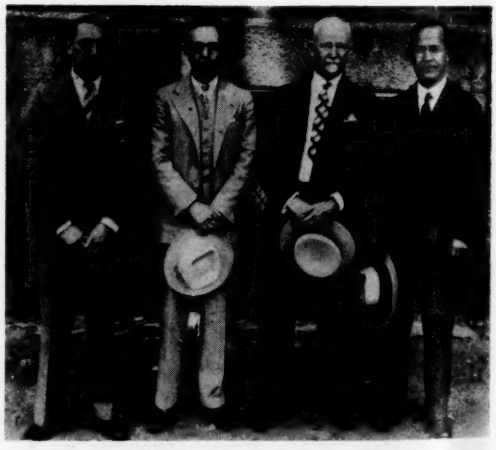
Irish, Scots, Welsh, and Germans besides the native English settled Lonaconing, but only the Scots and Germans remained.

By 1853, a railroad traversed the valley and the first newspaper was started in 1872. When the War between the States began in 1861, the late Dr. G. Ellis Porter made a public speech and thus originated the Home Brigade in defense of the Union. Not one man from Lonaconing went into the Confederate army.

Today Lonaconing has a population of 2,289.

In one hundred and twenty-one years, eighteen doctors have practiced in Lonaconing. While Dr. John A. Kolmer, world renowned immunologist was born in Lonaconing, he never practiced in the town. He is Emeritus Professor of Medicine at Temple University and the author of many books.

Physicians who have practiced in Lonaconing since its beginning are: Doctors James O. Bullock,



—THIRTY PLUS YEARS AGO—
(photo about 1927)
Drs. James A. Weinberg, E. David Weinberg, Minor Gibson Porter (deceased) and John A. Kolmer.

Arnold F. Gerstell, Herman, Henry M. Hodgson, Rosen Hunt, George G. Richards, J. D. Skilling, W. Q. Skilling, E. Don Taylor, Gabriel Ellis Porter, John M. Williams, G. H. Marmion, D. W. Williams, Michaels, Wundruh and Leslie R. Miles.

The present lone practicing physician is Dr. Leslie R. Miles and Lonaconing is badly in need of at least two more.

"For out of the old fieldes, as men saithe,
Cometh all this new corne fro yere to yere!
And out of old bookes, in good faithe,
Cometh all this new science that men lere.'

Chaucer

PERSONALS

Memorial Hospital, Cumberland, received an electrocardioscope from the local County United Fund sponsored by Dr. Wyllis Royce Hodges, President of the staff.

Dr. Raymond W. Reeves, Westernport, returned to civilian practice from the United States Army Air Force.

Drs. Lewis and Elizabeth Brings, of Cumberland, and their daughters, Marlene and Constance, have returned from a trip to the Great Smokies. Entering the Shenandoah forest at Front Royal, Virginia, they motored and camped along the trail through Virginia, North Carolina and Tennessee. Their biggest feat was to climb Mt. La Conte, which is over three thousand feet high.

Dr. and Mrs. James T. Johnson, Jr., of Cumberland, announce the birth of a son in the month of August.

FREDERICK COUNTY MEDICAL SOCIETY

LOUIS R. SCHOOLMAN, M.D.
Journal Representative

SOCIETY NEWS

The Society took its annual summer siesta; no meetings were held in July and August. No doubt the freshening Fall winds will arouse the Body in time to meet in September.

* * * * *

AIR FORCE MEDICAL SERVICE OFFERS RESIDENCY TRAINING IN GENERAL PRACTICE

The Air Force needs general practitioners. Approved residencies in General Practice are now available.

The Air Force General Practice Residency Program provides one year of training in medicine, including pediatrics and psychiatry, and one year in surgery—including traumatic surgery, fractures, obstetrics and gynecology.

It is anticipated that programs will be approved by July 1959 in hospitals at Andrews Air Force Base, Washington, D. C.; Keesler Air Force Base, Mississippi, and Wright-Patterson Air Force Base, Ohio.

For further information contact: The Surgeon General, Headquarters, USAF, Washington 25, D. C.

Book Reviews*

Aids to Materia Medica and Therapeutics, by J. W. Hadgraft, M.D., Fifth Edition, published by Bailliére, Tindall, and Cox; London, 1957.

This fifth edition was published in an effort to come abreast of the advances made in the study of pharmacology and chemotherapy in the last seven years. The author has attempted to present to the medical student "the way in which drugs are formulated for use in therapeutics and the alternative preparations available."

The official substances are described in alphabetical order. Each medication or group of medications is defined briefly. The range of dosage, the route of administration and the available forms are listed.

Although the goal of such a book is lofty and either beyond reach or, if attained, quickly lost by the relentless passage of time, Mr. Hadgraft has presented an economical, remarkably inclusive volume which is small enough to be carried in a student's or practitioner's bag. The use of British nomenclature should present no difficulty.

W. C. E.

Pharmacologic Principles of Medical Practise, Fourth Edition, By John C. Krantz Jr. and C. Jelliff Carr. 1313 Pages, The Williams and Wilkins Co., Baltimore, 1958. Price $14.00.

This book is devoted to the application of pharmacological principles and knowledge to the clinical practise of medicine. The text is well arranged; it proceeds in orderly fashion from the initial section on "The History of Pharmacology, Pharmacological Principles and an Introduction to Drugs" to the final section on "Allergy and Miscellaneous Drugs." The reviewer heartily endorses the author's definition of dosage—"It is enough . . . to accomplish the therapeutic aim intended." The section on the Central Nervous System contains an excellent discussion of the pharmacology of alcohol.

As could be expected in a work of this broad scope some omissions were noted. For example, in Chapter 2 on the Historical Development of Pharmacology, the discovery of insulin is not mentioned. In the discussion of "Miscellaneous Hypnotics" in Chapter 29 the "therapeutic uses of paraldehyde" on page 624 leaves much to be desired.

Two new chapters have been included in this edition. "The Use of Drugs in the Treatment of the Mentally Ill" starts with a basic discussion of brain metabolism and the drugs which effect it. The ataractic drugs are considered in a thorough and systematic manner. A second new chapter is devoted to "The Local Use of Drugs in Ear, Eye, Nose and Throat Practise."

The book is enthusiastically recommended both as a text book of pharmacology for the student and as a reference book for the practising physician.

L. P. G.

A Method of Anatomy, Descriptive and Deductive, by J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin. $11.00), The Williams and Wilkins Company, Baltimore.

The Sixth Edition of this publication is completely revised and contains much new material. The book is meant to be a working instrument designed to make anatomy rational, interesting and of direct application to the problems of medicine and surgery.

J. S.

Cold Injury, Ground Type, By Tom F. Whayne, M.D., Michael E. DeBakey, M.D., The Medical Department, U. S. Army, price $6.25.

This is one of the most comprehensive volumes on the ground type of cold injury that has ever been published. If it is carefully studied, and if the results of the observations of the United States Army in World War II are properly read and the facts retained, there should be no similar injuries in future wars.

J. S.

Twenty Thousand Nurses Tell Their Story, by Everett C. Hughes, Helen MacGill Hughes and Irwin Deutscher. Philadelphia, J. B. Lippincott Co., 1958, 280 pp. $5.75.

A candid and authoritative analysis of just where the nursing profession stands in America today is of importance to all who are concerned with providing nursing care for the people of this country. *Twenty Thousand Nurses Tell Their Story* provides just such an analysis, critical but warmly human.

This book is a sociological study of the nursing profession based upon a series of studies of nursing functions sponsored by the American Nurses' Association over a period of five years. Some twenty thousand nurses, as well as doctors, patients and the man-in-the street were questioned. Nurses who were observed were both professional and non-professional, employed at all levels, in all sizes and types of hospitals, and in non-institutional agencies.

* The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

In their analysis the authors consider: who nurses are; attitudes and expectations of student nurses; nurses' roles and relationships with patients, doctors and others; the influence of the "home guard"; classification of nurses into "the professionalizer," "the traditionalizer" and "the utilizer"; the effects when responsibility and power are not in balance.

Following are some excerpts from the introduction:

"We might call the nursing profession a 'more so' case; it is like many other occupations, only more so. That is what gives the study of nursing a wider interest. The things that are happening in it and to it are of the same order as those in many other occupations but are more strikingly visible."

"The name 'nurse' itself is historic, as are the uniform and the badge. But almost at the moment when, through licensing laws, the name Registered Nurse came to have clear and commonly recognized meaning, changes in medical technology and the organization of hospitals led to redistribution of the nurse's work so that it is no longer clear exactly who may be called Nurse."

"Certainly also, the standing of the nurse is changing; not in some simple way that affects all nurses equally but in complicated ways which reflect the demand of the modern medical establishment for a great variety of functions and for a corresponding variety of people to perform them."

"The medical division of labor will continue to become greater, in that there will be more and different kinds of things to be done and in that the health institutions will be more complicated and require different kinds of people to operate them. In the course of this, physicians will learn more new techniques and will continue to reorganize their work by delegation to others, especially nurses, of many tasks and responsibilities. . . . The doctor will keep control over the economy of his time by continuing such delegation, although, naturally enough, he probably will continue to complain that the woman to whom he delegates work and responsibility is not always there when he wants her on a moment's notice. The physicians' work will become more and more difficult and demanding; he will ask for help and will get it—and probably have to pay for it in changes of behavior and position which he will not especially like.

"There will be more work for nurses to do, and more and more kinds of nurses will be required to do it. Some difference of opinion about the solution of the many resulting problems will be chronic, but people will continue to work at them. Research will be part of the effort."

When the House of Delegates of the American Nurses' Association in 1950 decided to finance studies of nursing functions, it was with the expectation that the information obtained would provide a basis for improvement in the quality of patient care. *Twenty Thousand Nurses Tell Their Story* is more than an analysis of the nursing profession today. It points out implications for the nurse, for her relationships with others with whom she works, and for the future pattern of nursing.

Handbook of Treatment of Acute Poisoning. By E. H. Bensley and G. E. Joron. 212 pp. E. & S. Livingstone, Ltd., Edinburgh and London, 1958. The Williams & Wilkins Company, Baltimore, exclusive U. S. agents.

This book gives, in outline form, a guide to treatment of acute poisoning. The emphasis is on emergency measures. Physicians with no special experience in toxicology, when called upon suddenly and unexpectedly to treat cases of acute poisoning, can benefit tremendously by referring to this book. The material is nicely divided into two sections; the first contains a discussion of the principles, general plan and methods of treatment of acute poisoning. A thorough grasp of the contents of this section will enable the physician to treat acute poisoning cases without knowing the exact identity of the poison involved. The second section contains descriptions of important types of acute poisoning and the treatment to use. Most of the substances dealt with are relatively common causes of poisoning. Frequency of occurrence has been the main basis of selection. Finally, at the end of the book, the contents of an emergency antidote kit are listed.

Outlined in this text is a step by step procedure to be followed in the management of any poisoning case. Principles of treatment include prevention of further exposure to the poison, keeping the airway clear, control of respiratory depression, shock, convulsion, and secondary infection, and removal and inactivation of the poison.

The general plan of treatment is in two sections. The first—treatment before arrival of a physician—is intended to provide the physician with information that will aid him in prescribing over the telephone. The physician will then go to the patient's home, or the patient will be brought to his office or to the hospital. Thereafter, treatment will proceed under medical supervision. The second part of the plan of general treatment consists of the steps to be taken after the arrival of the physician.

The concise presentation of the information in this book makes it a very valuable reference text for all in general practice, in fact, for any physician who may be asked for advice in the management of a patient who has been poisoned.

J. R. G.

Dietary Prevention and Treatment of Heart Disease. By John W. Gofman, M.D., Alex V. Nichols, Ph.D. and E. Virginia Dobbin, Senior Dietician. Published by G. P. Putnam's Sons, New York. Price $3.95.

The merits of this book depend on whether or not the views of Gofman and his associates are correct. It is their claim that disturbances of the blood lipids, particularly lipo-proteins, are involved in the genesis of arteriosclerosis of the coronary vessels and that this process can be interrupted by dietary means.

The book, written mainly for non-medical readers, makes easy reading. It begins by describing the coronary circulation, its importance, and how it is affected by arteriosclerosis. The development of modern theories from the original concept of Virchow, that abnormal fatty substances in the blood may infiltrate the coronary lining, is traced. The authors outline in simple terms their own view that determination of the lipo-protein level is the principal means of detecting which individuals are prone to arteriosclerosis. The roles of saturated and unsaturated fatty acids, animal and vegetable fats, carbohydrate and protein are discussed. The remaining three-quarters of the book are devoted to prophylactic dietary schemes presented in relatively simple form and in great detail.

The book is an excellent guide if the authors' theories are correct. At present, however, there is no general acceptance of their conclusions and one wonders if they are really justified in warning the public to undergo the inconvenience of blood testing and, when indicated, to adopt the recommended dietary alterations. G. C.

Basic Cardiology. T. E. Gumpert, M.B., Ch.B., F.R.C.P. (Lond.) Williams & Wilkins Co., Baltimore.

This book consists, with the exception of the first and last chapters, of lectures given in the author's course at Sheffield University. They are not intended to take the place of a standard text-book on cardiology. The purpose of the publication of this book is to "stimulate the earnest student, and even the postgraduate, to a fuller understanding and a more rational practice of one of the most satisfying branches of clinical medicine."

Pye's **Surgical Handicraft,** edited by Hamilton Bailey, F.R.C.S., F.A., C.S., F.R.S., published by Williams & Wilkins Company, Baltimore.

This is a seventeenth edition and brings this compendium up to date. It is an all-round good text for reference. It is made more valuable by copious illustrations—of which there are 860—some in color.

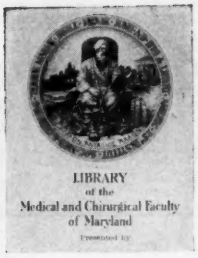
* * * * *

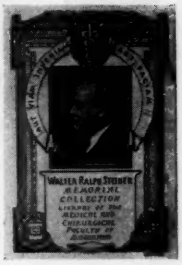
UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE ANNOUNCES SECOND PINCOFFS LECTURE

Monday, December 15, 1958

The School of Medicine of the University of Maryland announces that the second annual Pincoffs lecture will be delivered by Dr. George Thorn, Hersey Professor of Medicine, Peter Bent Brigham Hospital, Boston. The topic will be "Adrenal Hyperfunction—Clinical Studies." The lecture will be given in Chemical Hall in the old medical building at Lombard and Greene Streets, Baltimore and will start at 8:15 p.m.

This lectureship was established in 1957 by the faculty of the School of Medicine to honor Dr. Maurice C. Pincoffs and commemorate his thirty-five years as Professor of Medicine and Preventive Medicine.





Library

Louise D. C. King *Librarian*

"Books shall be thy companions; bookcases and shelves, thy pleasure-nooks and gardens." *Ibn Tibbon*

LIBRARY HOURS: Monday through Friday 9 a.m. to 5 p.m. Saturday 9 a.m. to 1 p.m.
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"Much study is a weariness of the flesh"
Ecclesiastes 12:12

BOOK SELECTION

"Study is a weariness of the flesh" only when interest is lacking. A book, well written and presenting facts lucidly, should be a pleasure and a stimulation to the reader but, because of the enormous output of textbooks and monographs on medicine and its related fields, the average doctor becomes discouraged before he even begins. His time is limited, he is not able to use the facilities available to the Library Committee and often puts too much faith in the agent's high powered sales talk or the publisher's summaries.

The Library Committee has difficulty in making a choice from the flood of publications crowding the market and although their choice need not be quite so selective as an individual's, it is their duty to avoid cluttering our shelves with works of no immediate or future value. Bearing this in mind, we are sending to specialists in various fields who are not members of the Library Committee, a list of new titles, requesting their aid in checking the list and asking them to make suggestions of their own of titles for purchase.

In this way, we hope to keep you abreast of medicine as it is today, without your becoming lost in the morass of new books.

We should like to appeal to each and every one of you who has read so far, to send in YOUR suggestions. When you have read or heard of a good book, jot down the name of the author, the title, date of publication and if known, the name of the publisher and send them to the Library as a possible purchase item, thereby doing your bit toward making your Library as you would like it to be. All such books, if desired, will be sent to the member recommending them before being put on the shelves. If the book is already here, we will mail it to you free of charge. All postage to borrowers is paid for by the Library, the borrower paying only for their return.

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SOCIETY OF PATHOLOGISTS INC.
PAUL F. GUERIN, M.D., *President* ROBERT D. SOLOMON, M.D., *Secretary*
Sinai Hospital, Baltimore 5, Md.

THE ONE-PINT TRANSFUSION

The collection and administration of blood carries much emotional impact for a patient, his relatives and sometimes for his physician. Such emotional reasons, unvoiced and probably not recognized, appear to play a part in the "justification" for some one-pint transfusions.

There are, of course, medically valid reasons for the single or repeated one-pint transfusion; however, many such transfusions are unjustified. Among the latter are those given "to get the patient out of the hospital and back to the family more quickly", "to give the patient a tonic", and "just to hurry things along". These objectives are commendable but they do not justify transfusion especially when balanced against the serious consequences which may result.

Consider these dangers of transfusion. In a recent survey of unselected patients, 1 per cent had abnormal antibodies to red cells. Two to five of each thousand persons transfused develop homologous serum hepatitis. Clerical errors leading to transfusion of the wrong blood account for some severe and often fatal hemolytic reactions. Consider also the young female immunized by transfusion (often enough to antigens other than Rh) and possibly deprived of future children.

Borderline or mild anemias are sometimes treated by the one-pint method as an adjunct to other therapy. It has been shown in the patient with iron-deficiency anemia that a rise in hemoglobin can be obtained in one week with iron therapy, equal to the rise expected from one pint of blood. In such cases, blood transfusion may be a poor source of iron, a wasteful source of protein and a risky pathway to health.

The one-pint transfusion also occurs in the operating room. Too frequently after surgery all except one unit of blood is returned to the bank. This pint is usually given as a prophylactic transfusion begun at the time the incision is made. The patient is exposed not only to the risks of surgery, but also to the hazards of transfusion multiplied by the fact that anaesthesia may mask a serious hemolytic reaction. The surgeon will not undertake an elective operation without adequate preparation of the patient. One reason for this preparation is to keep the unexpected from happening. Good judgment demands that similar caution be exercised in regard to the one-pint transfusion.

The Heart Page

Gordon Walker, M.D. – *Coeditors* – Robert Singleton, M.D.

A SERVICE OF
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JANUARY 15, 16, 17, 1959

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Dr. Caroline Bedell Thomas
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Sessions on

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Blue Cross - Blue Shield

MEDICINE'S SHIELD OF SECURITY

DENWOOD N. KELLY*

What are the prospects for the independent, private practice of medicine in the United States?

Any realistic attempt to answer this question must take account of many factors, some of them ominous indeed. In a letter to the Editor of the New England Journal of Medicine for April 3, 1958, Dr. Charles H. Bradford, a Boston physician, assesses the medical profession's basic socio-economic problems in clear and candid terms.

"On one side," he points out, "we find ourselves threatened by State and Federal programs, already operating on a large scale and eagerly seeking further enlargement. On the other side, we contemplate the ugly possibility of domination by pressure groups.... In front of us we face the inescapable and insurmountable wall of rising costs, in which doctors' fees play only an insignificant part as compared to the staggering costs of hospitalization. Behind us we hear the hue and cry of an increasingly hostile public opinion, unleashed and led on by demagogues who clamor for the priceless gifts of life and health at bargain rates and at the taxpayers' expense...."

As a primary weapon against these encircling troubles, Dr. Bradford points to Blue Shield, "one of the few constructive forward steps taken by the medical profession in the last twenty years..." which, he believes "may represent one of our few hopes for survival as a self-determining profession."

Blue Shield, the doctor asserts, has fulfilled its original purpose, namely, "to convert a system of postponed payment... into a streamlined system of prepayment..." to the benefit of both the people and the profession. But now, he suggests, Blue Shield is fulfilling an even more important function. It has given "what we as a profession never possessed before...an intelligently coordinated and professionally controlled corporate body through which we can bargain with the public and they with us."

Dr. Bradford feels that "if we continue to administer Blue Shield wisely, it may be all that we need to retain our professional independence." But to do so, he warns, "we must be willing to give as well as to receive—to regulate our practice and our fees so as to preserve our freedom from being regulated, and to grant the public as many benefits as we hope to assure for ourselves.... We, as doctors and as members of the medical society, are the managers of Blue Shield. Its success depends on us. Its failures remain our responsibility."

And, the doctor concludes: "We must grasp the larger significance of the splendid organization that we have built.... We must stand behind it... not for selfish gain, but to share with the public a mutual service and benefit. In this way, and in no other, can the medical profession preserve its time-honored status as an altruistic body of men, free from the sordid controls of politics or commerce, devoting itself wholeheartedly to scientific and humanitarian tasks."

The Editor of the Journal, in his comments on Dr. Bradford's letter, also made several points worthy of repetition. Pointing out that ever-changing world conditions are at present going through a series of major, critical alterations, he said, "as changes come to this country medicine must not always be found fighting a delaying action, especially in view of its relations with the public, which are perhaps at one of their lowest points in recent history. Whatever course the nation takes in its social, political, and economic evolution, the medical profession, for its own salvation, must lead in the development of constructive plans for the delivery of medical service under the new order, whatever it may be.... Blue Shield remains the doctor's own generally accepted agency through which he can deliver what a large percentage of the public seems to want—a plan for prepaid medical care—and through which he can keep an intelligent, constructive control over his hire and the conditions under which the care is provided. His success in maintaining the confidence of his public and in regaining it, where it has been lost, will depend on what he gives and not on what he gets. The practice of medicine is still, in its fundamental aspects, a service as dedicated as the ministry, and those who base their success on their income are not truly in that service."

* Assistant Director, Maryland Medical Service, Inc.

Health Departments

BALTIMORE CITY HEALTH DEPARTMENT

Another Look at the Lung Cancer and Smoking Matter

In an earlier paper entitled "The Nature of the Evidence of the Association of Smoking and Lung Cancer" which appeared in the January, 1955 issue of the *Baltimore Health News*, the following conclusions were reached by Dr. Matthew Tayback, Assistant Commissioner of Health for Research and Planning and Mr. Todd M. Frazier, Director of the Bureau of Biostatistics in the Baltimore City Health Department: (1) There is no doubt that a clearly proven *association* exists between excessive cigarette smoking and death from lung cancer, and (2) it is not clearly established that excessive smoking *causes* lung cancer.

During the past three years, as additional evidence has been produced by the U.S. Public Health Service, the American Cancer Society, the British Medical Research Council, and by other investigators, and in view of the consistency of the evidence, the Baltimore City Health Department is now convinced that: (1) It is highly probable that the smoking of cigarettes by itself, especially where more than one pack a day is smoked by a given individual, leads to a marked increase in the risk of lung cancer; (2) the increase in risk is proportional to the number of cigarettes smoked daily; and (3) it is also highly probable that excessive cigarette smoking is one of the more important causes of lung cancer.

It is recognized that this controversial subject requires further study. For instance, it is pertinent to determine whether smokers and non-smokers exhibit differences in measurable genetic, psychological, and sociological characteristics. In this connection, an investigation has been started by the City Health Department's Bureau of Biostatistics to ascertain whether the blood type of smokers differs from that of non-smokers. Another moot point is the influence of inhaling on the matter of lung cancer risk. However, this further activity need not deter a program of education designed to bring to the public and especially to teen-aged children, the known facts concerning the lung cancer-smoking issue.

The City Health Department is now considering the advisability of studying the smoking habits of high school students, particularly with a view towards understanding the motivation of such young people in starting the habit of cigarette smoking. It is fully appreciated that a major block, perhaps the chief obstacle to any success in efforts to reduce the percentage of teenagers who begin cigarette smoking is the overpowering influence of the example set by older persons.

Huntington Williams, M.D.

Commissioner of Health

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Ancillary News

NURSING SECTION

M. RUTH MOUBRAY, R.N., *Executive Secretary,*
Maryland State Nurses Association
Journal Representative

FOR THE INFORMATION OF PHYSICIANS

The American Nurses' Association and several state nurses associations have received inquiries regarding the American Registry of Doctor's Nurses which has been soliciting membership through direct mail communications to doctors, nurses, and others in the health field.

Should you receive such a communication, you might be interested in information on the organization recently made available to state nurses associations by ANA.

The ANA states that according to all available information, it appears that the organization is a commercial enterprise which is not recognized by professional associations in the health field.

The office of the Attorney General of the State of Florida, where the organization has maintained a mailing address, has released an opinion that the operation of the American Registry of Doctor's Nurses was in conflict with, among other things, the law regulating the practice of nursing in Florida. The organization then announced that it intended to discontinue its operation in Florida. To date, ANA has had no reports of mailings from addresses in other states.

ANA also reports that the Certified Nurses Aides of America, a similar organization using the same mailing address, also appears to be a commercial enterprise which is not approved by any recognized nursing or hospital organization.

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THREE CONFERENCES IN BAHAMAS SCHEDULED FOR THIS WINTER

The Sixth Bahamas Medical Conference, November 28, until December 18, 1958.
The First Bahamas Surgical Conference, December 29, 1958 until January 17, 1959.
The Serendipity Session, January 18 until January 31, 1959.
All Conferences will be held in the British Colonial Hotel, Nassau.
For further information contact Dr. B. L. Frank, Organizing Physician, 23 East 79th Street, New York 21, N. Y.

Woman's Auxiliary Medical and Chirurgical Faculty

MRS. DAVID S. CLAYMAN, *Auxiliary Editor*

SEMIANNUAL MEETING—SEPTEMBER 12, 1958

MRS. OTTO C. BRANTIGAN

The weather was just perfect—really made to order for the Semiannual Meeting of the Medical and Chirurgical Faculty and the open Board meeting of the Woman's Auxiliary at Ocean City on September 12th.

Well, friend, if you were not there you had better think about it! Your husband must be interested in what is going on in the State medical society. Perhaps he doesn't realize that he is most welcome to sit in on the meeting of the House of Delegates, which is the governing body of the association. For those actively engaged in the practice of medicine in Maryland, this meeting should be most interesting. Maybe he doesn't know he is welcome! Now, why don't YOU tell HIM?

Preceding the Board meeting the Hospitality Committee provided coffee and buns which really hit the spot, and the "How-de-do's" of old friends meeting again and new friends being greeted for the first time got the Auxiliary meeting off to a good start. All the ladies were urged to stay for the Board Meeting.

Immediately after the pledge of loyalty our speaker for the day, Dr. John M. Welch (Colonel, U.S.A. Retired) was introduced by our President, Mrs. E. Roderick Shipley. Colonel Welch is at present Chief of the Medical Services for Civil Defense. His topic was "The Present Status of Civil Defense in Maryland." He explained to us why plans for Civil Defense have seemed so confused, but he assured us that the apparent confusion has been due to reorganization to handle disasters which could result from the development of new weapons. The Civil Defense Department now has plans for setting up emergency stations and hospitals where studies have shown that they will be needed. He stressed the fact that there is still a great need for volunteer workers and expressed the hope that we would not lose interest in Civil Defense.

The Minutes of the Annual Meeting and of the previous Board meeting were read and approved. Following the Minutes, reports were heard from various committees which participated in the workshop which followed the last Board meeting.

Our meeting adjourned at 11:40 a.m. and while the men had their scientific meeting the ladies had time to relax in the sun.

At 2:30 p.m. the crowd gathered on the beach in front of the Commander Hotel for one of their famous clambakes. For those who have had the pleasure of participating in one of these feasts, I needn't say more. For those who have missed them in the past, I merely say—you shouldn't miss again! The seafood (clams on the half-shell, baked clams, clam chowder, deviled crabs, steamed crabs, lobster) was a real treat. In addition to the seafood there was delicious southern fried chicken, tomato juice, corn-on-the cob, iced tea, and sweet, red juicy watermelon.

The perfect day was concluded by an evening of dancing which was provided by the Medical and Chirurgical Faculty.

Now! Have you thought of a better excuse for spending a beautiful late summer day at the seashore?

"DOCTOR SAVERS—"

MRS. GEORGE H. YEAGER

This column will be devoted to helpful hints "on how to save the weary doctor. As doctors' wives we are all too helpless at times to offer any physical surcease to overworked husbands such as a moment spent sitting down or resting, any privacy, any vacation from the telephone, silence, or even time for a shower. She can only offer mental and spiritual support—understanding.

Perhaps doctors and their wives would mail in to "Doctor Savers" their own pet solutions. For example, young physician's wife to older, "My husband talks to me so little that I can barely ask him when he is available to go out to dinner". Older wife with final patience "Don't complain, my dear, mine has hardly spoken to me for years at home. It's not that he dislikes me, he is just bled white of talk by patients."

One doctor's favorite formula for a doctor saver is to be allowed to come home and stretch out on the sofa for ten minutes in complete silence and privacy, speaking to no one—then, after that, bring on all those problems of the children and the leak in the roof.

Send us your favorite "Doctor Saver," Doctors and Doctors' wives!

A THANKSGIVING MESSAGE

MRS. E. RODERICK SHIPLEY

"Not for myself, I make this prayer
But for this race of mine
That stretches forth from shadowed places
Dark hands for bread and wine."

Pagan Prayer
Countee Cullen

Thanksgiving is perhaps the time for us of the family of medicine to stop and think of the things for which we should be thankful.

The half century just past has seen greater scientific advances than have ever been made in a corresponding period of time.

X-ray, vitamins, insulin, polio vaccine, cobalt therapy, virus antitoxins and improved equipment and techniques for general surgery are now in daily use.

Understanding of mental diseases has progressed phenomenally in our time and the use of new therapies is returning to society many patients who formerly would have been incurable.

Pediatrics is now rated as a science and geriatrics may soon reach that status.

Today is a single point in the stream of medical advancement. The past is behind us, the future is tomorrow. We should be proud of the past, cognizant of the present, and prophetically certain of the future.

With *our* heritage, let us give thanks that as a corollary to the medical profession, with its accomplishments and prestige, we have been able to contribute as much as lies within our power. We have helped the medical schools, contributed to public education for better health, promoted safety, and enabled many to enter the field of medicine because of the financial assistance we have made possible. Last, but not least, let us be thankful for the knowledge, friendship and understanding that comes to us from a common purpose shared with our husbands.

Let us be thankful that we live in a free country—with freedom from fear, freedom from want, freedom of worship and freedom of speech. Now, add a fifth freedom, freedom in medical practice. Medicine, that has been shackled in many places in the world, is still free in America. We are privileged by the *four* freedoms to keep it that way and are thankful that this is so.

"Almighty God, Father of mercies, we, Thine unworthy servants, do give Thee most humble and heartfelt thanks for all Thy goodness and loving kindness to us, and to all men."

The Book of Common Prayer

Coming Meetings

BALTIMORE CITY MEDICAL SOCIETY

Friday, December 5, 1958 8:30 P.M. 1211 Cathedral Street

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ORTHOPEDIC SECTION

Tuesday, November 25, 1958 8:00 P.M. The Children's Hospital, Inc.

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OPHTHALMOLOGICAL SECTION

Thursday, December 4, 1958 6:30 P.M. Dinner Meeting, Johns Hopkins Club, Homewood

Dr. A. D. Ruedemann, Sr. of Detroit, Michigan will speak on

Techniques and Results of Intra-Scleral Implants

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CANCER SECTION

Wednesday, December 10, 1958 8:00 P.M. Veterans Administration Hospital, Fort Howard

Panel Discussion: "Management of Advanced Malignant Disease"

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PATHOLOGY SECTION

Monday, December 15, 1958 8:30 P.M. Sinai Hospital

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WOMAN'S AUXILIARY TO THE BALTIMORE CITY MEDICAL SOCIETY

Wednesday, December 3, 1958 12:00 Noon 1211 Cathedral Street

"Drug Addiction—A Medical Hazard" (Illustrated)

Speaker: John C. Krantz, Jr., Ph.D., Professor Pharmacology, Head of Department, University of Maryland School of Medicine.

Sandwiches and Coffee, 12:00 Noon.

POST FOR FUTURE REFERENCE

●

NARCOTIC PRESCRIPTIONS

A guide for physicians, dentists, veterinarians and pharmacists in the prescribing and filling of narcotic prescriptions in conformity with Federal and State of Maryland Laws.

1. Federal regulations classify all narcotics (and their preparations) into the following three classes and require each original package of a manufacturer or distributor to place the appropriate code letter on each label.

> A—Requires a written prescription.
> B—Oral prescription permissible.
> X—Exempt narcotic.

2. CLASS A NARCOTICS—WRITTEN PRESCRIPTION REQUIRED.

A. A written prescription must be presented before delivery of all narcotic preparations in Class A.

B. **A written prescription is required for:**

(1) Straight narcotics (includes salts and compounds)
   a. CODEINE (methylmorphine)
   b. DIHYDROCODEINONE
   c. DIHYDROHYDROXYCODEINONE
   d. DIONIN (ethylmorphine)—includes ophthalmic solutions.

(2) MORPHINE or any of its salts or compounds.

(3) COCAINE or any of its salts or compounds.

(4) OPIUM and its compounds; includes Pantopon.

(5) Synthetics and derivatives—ALONE or IN COMBINATION with other ingredients:
   a. DEMEROL (Meperidine, Pethidine)
   b. DILAUDID (Dihydromorphinone; Hymorphan)
   c. METHADONE (DOLOPHINE, Amidone, Adanon)
   d. Levo-Dromoran
   e. Metopon
   f. Isomethadon
   g. Methadol
   h. Nisentil (Alphaprodine)
   i. Thebaine
   j. Phenadoxone (Heptazone)
   k. Anileridine (Leritine)

3. CLASS B NARCOTICS—PERMISSABLE ON ORAL PRESCRIPTION.

A. Pharmacists must reduce oral prescriptions promptly to writing and file.
B. Practitioners must supply the same information as for written prescriptions.
C. Refills can not be authorized; a new prescription must be obtained each time.
D. Oral prescriptions are not limited to pre-manufactured preparations. Extemporaneous prescriptions are permissible when in conformity with these regulations.
E. The pharmacist is responsible for making certain that a drug or preparation furnished on an oral prescription is in fact authorized.

TABLE OF ORAL PRESCRIPTION PROVISIONS

DRUG	LIMITS	QUALIFICATIONS
(1) **Codeine** (methylmorphine) or any salt thereof	8 grains per fluid ounce or 1 grain per dosage unit	Codeine must be compounded with either: (a) One or more active, non-narcotic ingredients in recognized therapeutic amounts, (Example: Empirin with Codeine) or (b) an equal or greater quantity of any isoquinoline opium alkaloid or salt thereof (Example: Copavin).
(2) **Dihydrocodeinone** or any salt thereof (Dicodid, Hycodan, Hydrocodone, Mercodinone)	1-1/3 grains per fluid ounce or 1/6 grain per dosage unit	Must be compounded with either: (a) One or more active, non-narcotic ingredients in recognized therapeutic amounts (Example: Hycomine Syrup, Hycodan Tablets and Syrup, Orthoxicol) or (b) a four-fold or greater quantity of any isoquinoline opium alkaloid or salt thereof (Example: Tablet Dihydrocodeinone 4 mg. and Papaverine, 16 mg.)
(3) **Dihydrohydroxycodeinone** or any sale thereof (Oxycodone, Eucodal)	2/3 grain per fluid ounce or 1/12 grain per dosage unit	Must be compounded with one or more active, non-narcotic ingredients in therapeutic amounts (Examples: Percodan, Nucodan, Percobarb).
(4) **Dionin** (ethylmorphine) or any salt thereof	1-1/3 grains per fluid ounce or 1/6 grain per dosage unit	(a) Must be compounded with one or more active, non-narcotic ingredients in therapeutic amounts. (b) Plain ophthalmic solutions not permissible on oral prescriptions—other active ingredients must be present.
(5) **Isoquinoline Alkaloids** or any salt thereof	Oral provisions apply to any of these alkaloids **alone** or **in combination** with other active, non-narcotic medicinal ingredients. The following "isoquinoline" alkaloids are in current use: Papaverine, Narcotine (Noscapine), Narceine and Cotarnine (Styptol, Stypticin).	
(6) **Apomorphine** or any salt thereof	ALONE or in combination with other active, non-narcotic medicinal ingredients. It is not considered addicting and is not under international control.	
(7) **Nalline** (N-allyl-normorphine, Nalorphine) or any salt thereof	ALONE or in combination with other active, non-narcotic medicinal ingredients. It is not considered addicting and is not under international control. It is an antidote for poisoning by morphine, codeine, Demerol, etc.	

4. CLASS X NARCOTICS—EXEMPT NARCOTICS.

A. Exempt narcotics are not subject to the provisions of the oral prescription narcotic regulations, but prescriptions for Class X narcotics may, of course, be taken orally, reduced to writing and filed on the narcotic prescription file. Refilling, in good faith, is permissible and a record should be maintained of such refills on the prescription file.

B. The preparation shall contain active medicinal drugs other than narcotics in sufficient proportion to confer upon the preparation valuable medicinal qualities other than those possessed by the narcotic drug alone.

NOTE: These regulations are subject to change from time to time and inquiries should be directed to the Federal and State narcotic authorities.

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